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A PRELIMINARY REPORT OF SOME CASES OF PURPURA HÆMORRHAGICA DUE TO BENZOL POISONING.

By L. Selling, M. D.

(From the Pathological Laboratory of the Johns Hopkins Hospital.)

The cases which we have to report belong clinically to the group of purpuras; and from the mode of occurrence and from the results of the experimental work it is probable that they belong to the group of toxic purpuras. They occurred at a factory, in the suburbs of Baltimore, engaged in the manufacture of tin cans.

The factory is of fair size, employing about 325 hands and divided into several departments. The cases were not distributed uniformly throughout the factory, but were confined to one department, in fact, to one room of that department, the so-called "coating room." The only essential difference in the processes as carried on here and in the rest of the factory consisted in the use of a substitute for solder; a mixture composed of pure rubber, a rosin, and a coloring matter, dissolved in commercial benzol. Twenty-three hands were employed in the coating room; five men as machinists, four girls as inspectors, and fourteen girls, between the ages of 14 and 16, at the coating machines themselves. It is among these last that most of the cases, and all of the serious cases, occurred.

Their work was easy. One-half of them had simply to feed the can covers to the machines, which automatically carried them around and coated them about the margin with a thin layer of the pasty solution, whose constitution has been described, while the other half removed the coated covers. The covers were then piled in trays, inspected, and allowed to dry by the evaporation of the benzol. Ten gallons of benzol a day were used and allowed to evaporate in this room. In spite of the wide open windows and excellent ventilation—the cases occurred in summer—the space immediately about the coating machines was pervaded by a well-marked odor of benzol, while the same odor, much weaker, was noticeable throughout the rest of the room.

Case I.—M. W., white, female, aet. 14 yrs. Occupation: Factory hand.

The patient was first seen by Dr. Girdwood about June 1, 1909. At that time he was treating the mother for pneumonia and his attention was called to the daughter incidentally by her marked pallor and by the presence of a purpuric eruption. She was feeling perfectly well. Shortly after this she began to have hæmorrhages from the mouth and nose and she was advised by Dr. Girdwood to enter the hospital.

She was admitted to the Johns Hopkins Hospital June 28, 1909, complaining of "spots on body and dizziness."

Family History.—One uncle died of tuberculosis.

Past History.—Negative except for mumps, chicken-pox, and two attacks of measles. She had been working in the canning company for about four months at the coating machines. During this period she had no symptoms whatever and stopped work because of her mother's illness.

Present Illness.—The patient dates the present illness from the latter part of May, 1909, about one month before admission, when she first noticed blue spots on her arms and legs. These, she states, came out slowly and a few at a time. Shortly after this, she began to have bleeding from the gums, nose, and throat, one attack of epistaxis lasting two days. A few days before admission there was a very severe hæmorrhage from the throat, controlled only with difficulty by local applications. For the past week she has been in bed, the chief symptoms being weakness and dizziness. There have been no joint pains and no gastro-intestinal symptoms.

Physical Examination on Admission.—Patient is a well built, well nourished girl. Skin shows a marked waxy pallor and mucous membranes are very pale. Scattered over the arms and legs and, to a less extent, over the trunk are purplish-red to blue macules, 1 to 3 mm. in diameter and which do not change on pressure. Several fading spots are present on the face. On the right shin there is a large ecchymosis and a similar, but smaller one, on the left shin. The gums show slight bleeding, especially about the root of a broken tooth. They are not spongy. In the region of the left tonsil and the right anterior pillar and extending up to the uvula is a dark brown necrotic looking mass surrounded by an inflamed border. This was the site of a severe hæmorrhage which had been controlled by the application of ferrous sulphate before admission. No glandular enlargement.

Heart.—Negative except for systolic murmur over precordium, which was considered of hemic origin. Lungs negative.

Abdomen.—Liver edge easily felt 2 cm. below costal margin in right mammillary line; edge sharp and soft. Spleen not felt; dulness not increased. No œdema of legs.

Ophthalmoscopic Examination.—Moderate grade of neuro-retinal œdema. Fundi very pale and flecked throughout by great numbers of hæmorrhages, linear and round. These are especially numerous about the discs. The arteries are extremely pale. The veins are pale but relatively darker than the arteries.

For the first few days after admission the patient was listless, but, in spite of the marked anæmia, her condition seemed very good. There was a continuous, but slight, oozing from the mouth and throat, with an occasional expectoration of a blood clot. A few fresh purpuric spots appeared, some of them on the gums.

On July 3, the patient became very toxic. She was in a state of stupor from which she could not be aroused. The pulse was small and weak. Because of the seriousness of her condition, a transfusion of blood was attempted, following which the pulse became fuller and of a better quality and the mucous membranes of a slightly better color. There was, however, no change in the blood count.

July 4. The condition was much improved. The patient was mentally clear. The pulse slower and of better quality. The improvement continued until July 6, when she lapsed back into the previous toxic state. The respiration was rapid and labored, pulse rapid and of poor quality. She became steadily weaker until the exitus which occurred on that day.

During her stay in the hospital the temperature ranged from 99.8° to 104.6° F., the pulse from 108 to 165 per minute. Blood culture and Widal negative.

The urine was normal on admission, but following the transfusion showed albumin and a few hyaline and granular casts. There was no evidence of hemolysis either in the change in the color of the urine or in the appearance of jaundice.

Blood on Admission.—The examination of the fresh blood showed the red cells smaller than normal, pale and with very pale centres; moderate anisocytosis, but no extremely large or small forms. No poikilocytosis. Marked leucopænia with predominance of mononuclears.

	R. B. C.	W. B. C.	Hgb.
June 28	———	1,280	28% (Sahli)
July 1	1,090,000	480	11%
July 3	800,000	480	—
July 4	640,000	600	8%

In all the fresh smears examined, platelets were practically absent. Coagulation time 4½ minutes.

Differential count of smears made July 3: (Ehrlich's triacid stain.)

Polymorphonuclears	43%
Lymphocytes	41%
Large mononuclears	14%
Unclassified	2%

100 cells were counted. One megaloblast was seen; no normoblasts.

Contraction of the Blood Clot.—The blood, drawn from the ear and allowed to stand in a tube for 24 hours, showed some contraction of the clot and expression of the serum. The extent of contraction, however, and the amount of serum expressed were distinctly less than in the control tubes taken from normal patients.

Autopsy (abstract) by Dr. W. G. MacCallum. Anatomical diagnosis: Purpura hæmorrhagica (probably toxic); hæmorrhages in the skin, viscera, and serous surfaces; pallor of the organs.

Muscles a deep red color. Blood pale and watery. The heart muscle, and to a less extent, the liver, show some fatty degeneration.

Bone Marrow.—The bone marrow of the femur is fairly consistent; it is of a dull ochre yellow color with no abundance of blood supply. It does not look like a markedly hyperplastic marrow.

Microscopic Report.—Malpighian corpuscles of the spleen show areas of hyaline necrosis. Otherwise the findings simply confirm the gross description.

Bone marrow, microscopic examination by Dr. Boggs. Smear from the femur marrow (Romanowski stain) shows very few cells of any kind. The predominant cell is the normal red, which is pallid and slightly basophilic and shows no great irregularity. Leucocytes are extremely scanty. They are mostly of the lymphocytic or myeloblastic type. They are characterised generally by a very definitely reduced chromatin in the nuclei. Many nuclei show vacuolization and the cytoplasm is thin, fragile and poorly stained. One megalocaryocyte seen on fairly careful examination of the cover slip. Suggests an aplastic bone marrow.

Case II.—F. C., white, female, aet., 14 yrs. Occupation: Factory hand.

Admitted to the Johns Hopkins Hospital, July 6, 1909, complaining of spots on body and pain in the side.

The history, physical examination and the course in this case were so similar to those in the preceding that a detailed description is unnecessary.

She had been working in the factory for five months and had had symptoms for only one week. Previous to that she had felt perfectly well.

Blood Examination.—On admission the fresh blood showed the red cells of fair color with a very few pessary forms and a slight grade of anisocytosis. No poikilocytosis.

	R. B. C.	W. B. C.	Hgb.	
July 6	2,100,000	560	37%	(Sahli)
July 8	2,100,000	——	28%	
July 9	————	360	—	
July 12	1,150,000	140	15%	

Differential count of smears taken July 6. (Ehrlich's triacid stain):

Polymorphonuclears	16%
Lymphocytes	71%
Large mononuclears	10%
Eosinophiles	2%
Unclassified	1%

Only 100 cells were counted, owing to the scarcity of leucocytes.

A platelet count (Pratt's method), on July 9, gave 2500 platelets per cubic centimetre.

Blood taken from the ear and allowed to stand in tube for 48 hours showed absolutely no expression of serum. Control tubes from other patients reacted normally.

Autopsy (abstract) by Dr. Winternitz. Anatomical diagnosis: Purpura hæmorrhagica; hæmorrhages in the skin, viscera and serous surfaces; pallor of the organs; hyperplasia of the bone marrow; acute pleurisy.

The muscles are deep red in color, suggesting the presence of methemoglobin. Slight fatty degeneration of the heart.

The Bone Marrow.—The marrow of the long bones is deep red in color and shows marked hyperplasia. Its redness, however, is different from that generally seen in hyperplastic marrow. It is rather deeper, darker red, having the same hue described for the muscles. Smears taken from this marrow show not a hyperplastic, but an aplastic condition. The appearance closely resembles that of the bone marrow in Case I, with the exception that the reduction in the cellular elements had not reached quite so marked a grade.

CASE III.—A. N., white, female, aet., 14 yrs. Occupation: Factory hand.

Admitted to the Johns Hopkins Hospital, July 6, 1909, complaining of giddy spells and stomach-ache.

Patient had been working at the canning company for about three months, in the coating room. She had had symptoms for two months. These were chiefly anorexia, abdominal pain, vomiting and headache, and one or two fainting spells. The purpuric eruption in this case was very much less extensive than in the preceding cases. The general condition of the patient throughout her stay in the hospital was excellent. At no time did she show any alarming symptoms. She was discharged after six days and has continued well since.

The Blood Examination.—The blood examination shows the red cells practically normal in appearance.

	R. B. C.	W. B. C.	Hgb.
July 6	4,900,000	4,400	54%
July 10	4,740,000	5,800	50%

Coagulation time 6½ minutes.

Platelet Count.—104,000 per cubic centimetres. Clotting of blood and expression of serum diminished as in the other cases.

An examination of the other employees working in the coating room was undertaken, and four were found, two men and two girls, who showed a few purpuric spots. They were entirely free from symptoms. Their blood showed a slight grade of anæmia and a leucocyte count varying from 3900 to 5200 per cmm., that is, either subnormal or on the lower limits of normal. Control counts taken from those working in other parts of the factory gave the white blood cells 5200 to 6000, and red blood cells 4,000,000 to 5,000,000 per cmm.

A series of cases of almost exactly the same character was reported by Santesson,[1] of Stockholm, in 1897. The cases occurred at a factory in Upsala engaged in the manufacture of bicycle tires, and where benzol was used as a solvent for rubber. His cases showed a marked purpuric eruption and four of them terminated fatally.

So we have two series of cases of purpura, some purpura hæmorrhagica, and some purpura simplex, with all the clinical features of the idiopathic purpuras; even to the diminution in platelets and in the expression of serum from the clotted blood. Both series occurred in factories where benzol was used as a solvent for rubber. The cases were chiefly among young girls and the course was essentially chronic, although in the end the serious symptoms developed suddenly. It is noteworthy, also, that the disease progressed in spite of withdrawal from the influence of the toxic agent.

The most remarkable feature was the blood condition, and this can be best described as the picture of an aplastic anæmia. In common with the aplastic anæmia were, *First*, the presence of only slight changes in the appearance of the red blood cells. These showed, in our cases, only slight pallor and slight anisocytosis. *Second*, the absence of regenerative forms. In stained specimens of the two fatal cases, only one megaloblast and no normoblasts were found. *Third*, scantiness of platelets. *Fourth*, diminution of the granular types of white blood cells, with a relative increase in the mononuclear elements. In our Case I, polymorphonuclears were reduced to 43% while mononuclears were 55%. In Case II, the polymorphonuclears were 18% and the mononuclears 81%. *Fifth*, leucopænia. This reached a much more striking grade in our cases than in any of the reported cases of aplastic anæmia. In Case I, there was a count of 1280 cells on admission and later of 460 cells. In Case II, there were 550 cells on admission and on day of death the count had fallen to 140 cells per cmm. Similarly one of Santesson's cases showed, with a red blood count of 3,700,000, no white cells in the squares of the counting chamber, and only 5 to 10 around the margin of the disc.

In attempting to locate the source of the trouble, Santesson performed some experiments with benzol and benzene. Before discussing his work a few words with regard to these substances will not be out of place because a similarity of names has given rise to great confusion in the literature.

We have to consider three substances: Benzene, benzol, and benzine. *Benzene* is a chemical substance of definite and known constitution, C_6H_6 the ordinary benzene ring. The term *benzol* is used in two senses; first, as a synonym for

[1] Santesson: Archiv für Hygiene, Vol. XXI, 1897, p. 336.

benzene, and second, to designate a rather complex mixture of variable composition derived from the distillation of coal tar. I think we can make it plain by indicating the derivation.

The fractional distillation of coal tar yields primarily four products:

1. Crude naphtha distilling below 180° C.
2. Dead oils or creasote oils distilling from 180° to 270° C.
3. Green or anthracene oil distilling above 270° C.
4. Pitch, the residue.

It is from the first fraction that commercial benzol is obtained by redistillation and purification. The benzols of the market vary widely in composition according to the source from which they are derived and the extent of purification. In general, they consist chiefly of benzene and its homologues (Toluene, $C_6H_5 \cdot CH_3$, and Xylene, $C_6H_4 \cdot (CH_3)_2$) together with small percentages of carbon disulphid, pentane C_5H_{12}, thiophene C_4H_4S and its homologues, traces of water, acetylene and impurities of indefinite nature.[2] *Benzine* with which these two have been confused, is a derivative, not of coal tar, but of petroleum. It is composed of some of the lighter materials obtained from crude petroleum on distillation, and consists essentially of hexane, C_6H_{14}, and heptane, C_7H_{16}; usually it contains no benzene, C_6H_6.

Santesson tried the action of benzol and chemically pure benzene on rabbits by subcutaneous injection and by wrapping the bodies of the animals in cloths soaked with the material. In this way he produced chronic poisoning and in nearly all of his cases found at autopsy hæmorrhages in the pleura, lungs, mucosa of the stomach and intestine, and once beneath the pericardium. Similar hæmorrhages have been found by other workers with more or less regularity, in animals poisoned with benzol, and they have also been found in several cases of poisoning in man, that have come to autopsy. L. Lewin considers them characteristic.[3]

Subsequently, Santesson[4] performed further experiments based on the finding, in one of his fatal cases, of a marked grade of fatty degeneration of the organs. Although realizing that this may have been due to the anæmia itself, he attempted to reproduce the condition in rabbits, and succeeded. On the basis of the hæmorrhages and the fatty degeneration of the organs he concluded that benzol was the source of poisoning, and in as much as he could produce the same changes with benzene, that benzene was the essential toxic agent.

Our own work has been directed along somewhat different lines, although using the same substances. A priori, we felt that we could exclude the pure rubber, the rosin, and the coloring matter, which consisted simply of a clay highly colored with iron oxide, and turned our attention to the solvent. An analysis by Dr. C. Glaser, for the factory, revealed the presence of traces of carbon disulphid, thiophene, and of nitrobenzol; the last present in quantities of only one milligram per litre.

An analysis was made by Dr. Penniman, for the Maryland State Board of Health, by subjecting the entire mixture as used at the factory, to fractional distillation and dividing it into ten equal fractions. The first fraction started to distill at 70.6° C. and ended at 80.5° C. The next eight fractions distilled at 80.5° C., and the last was taken without further treatment. The boiling point of pure benzene is 80.5° C., so this formed more than 80% of the solvent. The first, fifth, and tenth fractions were injected into rabbits in doses suitable to produce chronic or subacute poisoning, 1 to 3 ccm. in olive oil. The effects were *identical* with the *three*, and, as far as our experiments are concerned, this excludes nitrobenzol from any share in the results. Nitro-benzol has a boiling point of from 203° C., to 209° C., so that any present in the original mixture would have been found *only* in the last fraction. Carbon disulphid in the quantities present could have played no part.

After a variable interval of from one to eight days the white blood cells began to diminish in number and in one case a count of 20 cells per cmm. was obtained. In the early stages of the poisoning there was an outpouring of a large number of abnormal elements among the white cells, either typical large lymphocytes with a large, deeply staining nucleus and a small amount of very basophilic protoplasm or closely related cells which showed a varying degree of basophilia of the protoplasm and irregularities in the nucleus. Myelocytes were not found except in one case. When the leucopænia reached such a low grade as above described, death occurred, even if the injections were stopped. The fall in the red blood cells was much less marked than the fall in the leucocytes; counts below 4,000,000 per cmm. being very uncommon. By carrying the poisoning over a much longer period, however, we succeeded in producing in one case a high grade of anæmia, with about 1,500,000 red cells. Many nucleated reds appeared in this experiment.

The autopsy findings in our experiments with respect to hæmorrhages were practically negative. In only one or two did we find any at all, and these were in the gastric mucosa. The bone marrow, in the examination of which we received much assistance from Dr. Whipple, showed striking changes. In cases that had rapidly developed an extreme leucopænia the marrow showed marked aplasia. The polymorphonuclears had been almost wholly swept out and a large percentage of the myelocytes showed marked signs of degeneration, while all forms of white cells were greatly reduced. The red cells, on the other hand, were practically uninjured, showing no signs of degeneration.

The bone marrow picture showed striking resemblances to the bone marrow of our two fatal cases. In both it was the white cells especially that suffered and of the white cells mainly the granular elements. In contrast to this is the picture obtained after toluene poisoning. In experiments carried on with this substance we found likewise a destruction of white blood cells, as evidenced by the primary reduction in their number and by the appearance of abnormal elements

[2] Allen: Commercial Organic Analysis, Vol. II, Part II, p. 176.
[3] Lewin: Münch. med. Wchnschr., LIV. II, 1907, p. 2733.
[4] Santesson: Skandanavisches Arch. f. Phys., Vol. X, 1900, p. 1.

among them in the circulating blood. In comparison with benzene, however, its action is feeble and the bone marrow is fully able to compensate the destruction. To meet the extra demands it must do extra work and, therefore, we find at autopsy a marked hyperplasia of the marrow, the fat being entirely replaced by an overgrowth of cells.

Experiments with chemically pure benzene yielded results identical with benzol, that is, leucopænia and aplasia of the bone marrow, and we have thus in benzene an intense poison for the white cells[5] of the blood. Its action on the red cells is much less marked. That there is some destruction here, too, is evidenced by an abnormal pigmentation of the spleen.

Finally the question arises whether we have actually demonstrated the toxic agent. The facts stand thus: first, the cases occurred only in that room of the factory where the benzol rubber solution was used. Second, similar cases have been reported in another factory where the benzol was used as a solvent for rubber. Third, the main features of the cases were purpura, anæmia, and leucopænia. Fourth, in the experiments with the benzol used at the factory we reproduced the leucopænia and in those directed toward that end, also the anæmia, that is two of the three chief symptoms; while others working with benzol have found evidence that it tends to produce hæmorrhages in different parts of the body. Fifth, benzene produces the same effect as benzol.

In closing we wish to express our thanks to Dr. L. F. Barker for permission to report these cases and to Dr. Boggs, Dr. Whipple, and Dr. Winternitz for their kind assistance, and to Dr. Price of the Maryland State Board of Health.

[5] Exposure to Roentgen Rays is likewise able to produce an enormous destruction of white cells.

ANERYTHRÆMIC ERYTHRÆMIA (?)

By ROGER S. MORRIS, M. D.,

Associate in Medicine, in Charge of the Clinical Laboratory, The Johns Hopkins University.

(From the Medical Clinic.)

During the last two years I have had under observation in the dispensary of the Johns Hopkins Hospital two interesting patients. Their histories were somewhat similar, and further similarity was strikingly present in their physical conditions. Without a blood count, the diagnosis of erythræmia was immediately suggested. But, as the following notes show, this could not be definitely established:

CASE I.—*Clinical Diagnosis.*—Anerythræmic erythræmia (?); epilepsy. A. H. (Dispensary No. E15,726), Austrian Hebrew, aet. 17, single, a clerk by occupation, was admitted to the Medical Dispensary, November 26, 1907, complaining of "nervous dyspepsia."

Family History.—(The following history was obtained at a later date with the aid of the patient's father:) Father and mother living; both complain of "stomach trouble" at times, but are otherwise well. A brother and a sister are well. No epilepsy or other nervous disease in the family. No tuberculosis.

Past History.—At the age of 1½ years the patient had "typhoid fever" in Austria; he was ill two months. At three he had "scarlet fever" and "diphtheria" in this country; good recovery. "Measles" at six. No other infectious diseases or fevers. No history of syphilis in parents. Patient denies exposure to venereal disease. No malaria.

Present Illness.—At the age of four the patient was seized with a general convulsion and loss of consciousness for 8 to 10 hours, the father says. Since then the attacks have been repeated at intervals varying from three to four days to a month. The patient says he is always warned of an oncoming attack by the fact that his "heart begins to hammer" for about a minute. His father says he has convulsions in some of his attacks, affecting the arms and legs, with grinding of the teeth. The patient "foams at the mouth and there is a rattling in the chest." The lips and face are always intensely blue during an attack; the patient says he has noticed blueness of the lips and face himself between the attacks, especially in the winter. He has bitten his tongue and passed urine and fæces involuntarily in some of the attacks. Following the attack he has headache and feels stupid for several hours. Milder attacks without convulsions are more frequent than the severe ones. In the former the same aura is noted; then the patient loses consciousness, and, if he happens to be on the street, he drops anything he may be carrying and may go four to six blocks without having any recollection of what he has done. Constipation has always seemed to bear a definite relationship to the attacks, the latter being more frequent when he is constipated. He frequently has slight vertigo and occasionally vomits. He has been constipated and has had to take laxative medicines since the attacks first began. The father has never noticed that the patient's face grew blue in cold weather, and the patient himself cannot say how long he has noticed it.

Status præsens.—No abnormality was noted on the day of admission, but this is probably accounted for by the fact that there was not sufficient time for a careful examination. I saw the patient for the first time December 28, 1907. At this time his lips were very cyanotic, his face and extremities less so. There was a diffuse cyanosis of the trunk of slight degree. The heart and lungs were negative.

The abdomen looked natural. The spleen was readily felt with the patient on his back breathing quietly; it extended about one finger-breadth below the costal margin, and its edge was firm and rounded. No enlargement of the liver could be demonstrated.

Blood examination made at this time showed 4,880,000 red blood cells and 89% hæmoglobin (Sahli).

The patient said he had never had malaria, but vertigo had troubled him for about five years.

March 4, 1908. Has had several attacks of petit mal. He has been taking laxatives and tonics.

R. b. c.	4,910,000
W. b. c.	12,000
Hb.	89% (Sahli)

Differential count of 628 white cells (Wilson's stain) gave: Lymphocytes 27.2%; large mononuclears and transitionals 3.3%; polymorphonuclear neutrophiles 60.3%; eosinophiles 7.9%; mast

cells 0.8%; neutrophilic myelocytes 0.16%; degenerated forms 0.3%. No alterations in the red cells were seen. Platelets were about normal in number. No malarial parasites were seen. No cause of the eosinophilia, such as skin disease, asthma, intestinal parasites, trichinosis, has been found.

March 7, 1908. *Blood* examined for the bands of methæmoglobin and sulph-hæmoglobin gave a negative result.

May 6, 1908. Cyanosis of lips and ears well marked. Hb. = 91% (Sahli). *Spectroscopic examination* of the blood again negative.

November 10, 1909. The patient was requested to return to the clinic for examination. He says he has felt better, on the whole, during the last eighteen months. The bowels have been kept open. He has had attacks of vertigo once in three or four weeks (evidently petit mal, according to the description given by his father), but in the last month two attacks have occurred. In January, 1909, the patient was in bed for a week and suffered with vertigo, drowsiness, and nausea.

The patient was shown by Dr. T. McCrae to a clinic for the students and the following is a brief abstract of his remarks: The patient is well nourished. The lips and ears have a bluish tint. Tongue clean. Mucous membranes good color. No jaundice.

Heart and lungs clear.

Abdomen tense. Spleen enlarged and firm, one inch below the costal margin. Abdomen otherwise negative.

In discussing the case, Dr. McCrae called attention to the fact that there was no evidence of leukæmia, malaria, lues, new growth, amyloid disease, typhoid fever, or splenic anæmia, and expressed the opinion that the case was probably an early erythræmia.

November 10, 1909. *Urine;* fresh specimen: Clear, yellow, acid, sp. gr. 1.013. No albumin or sugar. Urobilinogen—trace. Urobilin—negative with Schlesinger's test after addition of Lugol's solution. Sediment—none.

Blood.—R. b. c. 5,104,000; w. b. c. 5,600; hb. 107% (Sahli). Differential count of only 233 cells (Wilson's stain) gave: Lymphocytes 34.7%; large mononuclears and transitionals 4.7%; polymorphonuclear neutrophiles 56.2%; eosinophiles 1.7%; mast cells 0.82%; myelocytes 0.43%; degenerated forms 1.3%. No abnormalities in the red cells; platelets about normal.

November 22, 1909. No attacks of epilepsy in the last two weeks, but several spells of nausea and vertigo. Complains of burning of the lips. The patient has been taking Fowler's solution and calcium lactate.

Stool (fresh).—Normal color and odor; soft. Nothing abnormal was found on gross or microscopic examination. No parasites or ova.

Blood.—R. b. c. 5,000,000; w. b. c. 4,000; hb. 105% (Sahli). *Viscosity* of the blood (Dr. Austrian) = 5. Spectroscopic examination of the blood (kindly verified by Professor Abel) was negative for sulph-hæmoglobin and methæmoglobin. Differential count of 542 cells (Ehrlich's triacid stain) gave: Lymphocytes 25.2%; large mononuclears and transitionals 3.8% (of which about 1.1% were Rieder cells); polymorphonuclear neutrophiles 64.0%; eosinophiles 3.7%; mast cells 0.73%; myelocytes 0.73%; degenerated forms 1.6%. No nucleated reds, etc.

Dr. Forster, of the Phipps Dispensary, kindly examined the patient's lungs, before giving him the tuberculin test, and found the percussion note on each side good; breath sounds good. No râles heard anywhere, even after cough. Result: Normal lungs. The *tuberculin test* showed a positive von Pirquet, a negative ophthalmo-reaction after instillation of 1% and 5% old tuberculin, practically excluding an active lesion (Dr. Hamman).

Case II.—*Clinical Diagnosis.*—Anerythræmic erythræmia (?). K. H. (Dispensary No. E21,877). Russian Hebrew, aet. 21, single, a tailor, was admitted to the Medical Dispensary, April 1, 1908, complaining of "constipation and pain in the chest."

Family History.—Unimportant.

Past History.—Unimportant. Has always been well until the present illness. The patient denies venereal disease, and says that "whooping-cough" is the only disease he has had.

Present Illness.—Two years ago the patient was troubled with "giddiness in the head" and pain in the left chest in the region of the heart. He remained in bed for two months, recovered, and remained well for a year, when the trouble returned. The patient is not constantly ill, as he was at the onset. Some days he feels perfectly well; on others he complains of giddiness, nausea, and pain in the left chest. The vertigo comes on especially after meals, although he has noticed it at times before breakfast. Usually the symptoms are relieved by taking a dose of Epsom salts. The pain in the chest is quite constant, and at times extends into the back, shoulder, or arm. Deep breathing makes it worse. The patient is frequently a sufferer from headache "over the whole head." The appetite is poor. The bowels have been very constipated. The patient has no cough and has not lost weight. He sweats at night occasionally; does not feel feverish or chilly. At times he has aching pains in the legs.

Status Præsens.—The patient is poorly nourished and of slender build. There is slight general enlargement of the lymph glands. The tongue is moderately coated. Slight but definite cyanosis of the lips, ears, and nail-beds is seen. Eyes negative.

Heart and lungs are negative throughout on inspection, percussion, and auscultation Pulse 75. Temperature normal.

Abdomen looks natural. The aorta is readily felt and tenderness is complained of on palpating it. The edge of the liver is just felt. With the patient on his back the spleen is palpable and descends about 1 cm. below the costal margin. Splenic dulness begins on the 8th rib in the middle axillary line, and extends almost to the border of the ribs.

Dermatographia was very active. Knee jerks active.

The patient was given cascara and instructed to return a week later for blood examination.

April 8, 1908. *Blood examination* showed: R. b. c. 4,925,000; w. b. c. 8,600; hb. 99% (Sahli). Smears of the blood stained with Hasting's stain showed nothing unusual, but unfortunately a differential count was not made. Spectroscopic examination of the blood for the bands of methæmoglobin and sulph-hæmoglobin was negative.

(Subsequent attempts to locate the patient have failed, so that further study of the case was impossible.)

The notes of these two cases show their close parallelism. In neither could a history of malaria or syphilis be obtained to account for the splenic tumor; splenic anæmia did not exist, nor was there any evidence of amyloid disease. The blood examination excluded a leukæmia, and there were no grounds on which to base a diagnosis of Hodgkin's disease. There was no valvular disease of the heart, no pulmonary emphysema, to account for the cyanosis. Sulph-hæmoglobinæmia and methæmoglobinæmia, of which thirteen cases have been collected by West and Clarke (1) up to 1907, and another added by Russell and Leathes (2), were thought of as possible explanations of the cyanosis. The latter is not uncommon in these cases, but differs, according to Osler (3), in that it resembles more the ashen hue of argyria, as contrasted with the plethoric cyanosis of erythræmia. Splenic

tumor, which does not appear in the summary of West and Clarke, was found in both of my patients. Furthermore, repeated spectroscopic examinations of the blood in one case and a single examination in the other revealed only the bands of oxyhæmoglobin, even in concentrated solution, thus excluding sulph-hæmoglobinæmia and methæmoglobinæmia. Tuberculosis of the spleen, associated with splenic tumor, cyanosis, and polycythæmia, has been reported by Rendu and Widal (4) and by Moutard-Martin and Lefas (5); subsequent autopsies on cases of polycythæmia with splenomegaly have not shown a similar lesion. This possibility, though rare, must be kept in mind. Such a condition cannot be definitely excluded in Case II, though it is extremely unlikely to have been present. With the negative tuberculin test in Case I, confirming the physical findings, as it does, it is reasonably certain that an active tuberculous process is not at the base of the patient's condition.

Were it not for the normal blood counts, both of the cases reported above would be characteristic examples of erythræmia,[1] of which Parkes Weber (6) has recently published an excellent critical review. He defines erythræmia as "a disease or morbid condition characterized by well-marked persistent relative and absolute polycythæmia (increase in the number of the red blood corpuscles) due to an excessive erythroblastic activity of the bone marrow, which appears to be the primary morbid factor in the condition; it is characterized likewise by persistent increase in the viscosity and total volume of the blood, and usually by a cyanotic appearance of the patient and by enlargement of the spleen." This definition coincides with the prevailing opinion of the disease. Autopsies have revealed a red, hyperplastic marrow in the shafts of the long bones, and the disease has been looked upon as analogous to leukæmia; hence the name, erythræmia, proposed by Türk.

From Dr. Weber's definition, it is evident that the *sine qua non* for a diagnosis during life is a persistent increase in the number of red cells in the blood. This has been found almost constantly—though normal counts have been reported, generally coincident with a complicating (terminal) disease (sepsis, hæmorrhages)—in every case thus far recorded. Of the two remaining cardinal signs, cyanosis has been absent in some cases, while splenomegaly has been of more constant occurrence, according to Weber.

Microscopic examination of the stained blood in erythræmia has shown no constant abnormalities by which the disease can be recognized. Polychromatophilia is frequently seen. Nucleated reds are not uncommon, and are often present in considerable numbers. They have, however, been absent in some cases, even after prolonged search; but Boycott (7) expresses the belief that repeated examinations will reveal their presence in every case. From Naegeli's (8) careful analysis of the literature, it appears that myelocytes are of very frequent occurrence, and eosinophiles and mast cells are usually present in increased number. Myelocytosis is, as a rule, coincident with leukocytosis, but in one of Naegeli's patients the myelocytes amounted to one per cent with a total white count of only 6800. While we may encounter, then, pathological changes in the stained blood (polychromatophilia, normoblasts, megaloblasts (rare), myelocytes, eosinophilia, etc.), there are, nevertheless, no pathognomonic alterations, by which an absolute diagnosis of erythræmia can be made. The one characteristic blood feature thus far discovered is a polycythæmia with increase of viscosity. We are, therefore, not justified at present in making a positive diagnosis of erythræmia during life in the absence of an increase in the number of red cells.

In the history of leukæmia, there was a period, in which it was considered essential that there be a marked increase in the number of the white cells of the blood. As knowledge of the disease progressed and its morbid anatomy and hæmatology were more closely studied, cases of so-called aleukæmic leukæmia, *i. e.*, leukæmia with normal or subnormal white counts, were recognized. If the generally accepted theory of erythræmia be correct and we have in this disease the red cell analogue of leukæmia, it seems likely, to carry the analogy still further, that we will soon be able to recognize cases of erythræmia without increase in the number of red cells, or even with subnormal red counts. Were such a phase of the disease proven to exist, it would be designated properly "anerythræmic" erythræmia. That there is, or is not, an anerythræmic erythræmia may be demonstrated with our present knowledge (1) by clinical observation of cases, such as those recorded here, with a view to demonstrating a subsequent (or previous) polycythæmia, or (2) by the post mortem finding of the characteristic pathological changes in anerythræmic, but otherwise typical, examples of the disease.

In the cases reported above the suspicion has been strong that each represented an early (?) stage of erythræmia. The histories are quite suggestive—excepting the epilepsy in Case I, which could have no apparent bearing on a possible erythræmia—and physical examination demonstrated two of the three cardinal signs, *i. e.*, chronic cyanosis and splenomegaly. The usual causes of cyanosis and splenomegaly, excepting erythræmia, have been reasonably excluded, especially in Case I. The repeated finding of myelocytes in the blood of Case I is an abnormality which I have been able to explain on no other ground than that of undue activity (hyperplasia) of the bone marrow, the essential anatomical alteration of erythræmia. The eosinophilia noted early in Case I has been interpreted similarly, since no other adequate cause for it could be found. Eosinophilia of like significance is not uncommon in pernicious anæmia, for example, presaging, as it often does, a period of improvement with marked regeneration (and consequent hyperplasia of the bone marrow). For the reasons just given and by the process of exclusion, the provisional diagnosis of anerythræmic erythræmia has been made; for it seems improbable that the patients have been the subjects of a new, hitherto unrecognized clinical entity.

[1] Synonyms: Splenomegalic polycythæmia; myelopathic polycythæmia; polycythæmia with chronic cyanosis; erythrocytosis megalosplenica; Vaquez's disease; Osler's disease.

If the narration of these cases aids in any way in proving or disproving the existence of an anerythræmic stage in erythræmia, the object of the report will be attained.

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ON THE EXCRETION OF CREATININ IN THE INFANT WITH SOME NOTES REGARDING ITS OCCURRENCE IN AMNIOTIC FLUID.

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Until recently the opinion was held that the urine of infants did not contain any creatinin or that the creatinin did not occur constantly. It has been shown that this opinion was erroneous and that creatinin forms a regular constituent of the infant's urine. Moreover the amounts present, though smaller in proportion to the body-weight than in the adult, do not show much greater individual variations than are observed in the adult. The following table reproduces the result of the investigations obtained on infants which did not show any signs of ill health.

Table I.

Name of authors.	No.	Age of infants.	Weight in gm.	Total (24 hrs.) creatinin in mg.	Creatinin in mg. per kg.	Creatinin coefficient (Shaffer).
Amberg and Morril.	I.	10 days.	4033	26.82	6.71	2.5
	II.	10 days.	3463	26.92	7.78	2.9
	III.	14 days.	2723	27.05	9.94	3.7
	IV.	7 days.	3093	17.48	5.46	2.1
	V.	13 days.	3100	30.1	9.7	3.6
(avg. of 6 days)	VI.	1 mo.	4561	35.3	7.4	2.9
Funaro.	VII.	1½ mos.	3500	17.5	5.0	1.9
	VIII.	2 mos.	4000	21.0	5.25	1.95
	IX.	2½ mos.	4300	28.0	6.51	2.4
	X.	3 mos.	4380	31.2	7.12	2.6
	XI.	3 mos.	4000	30.4	7.6	2.8
	XII.	3 mos.	4800	42.0	8.75	3.3

With exception of No. XII all the infants were breast-fed.

The age of the infants in this table varies between seven days and three months. It seemed desirable to extend the investigation to the first days of life and the results are recorded in Table II.

Table II.

No.	Weight.	Total (24 hrs.) creatinin in mg.	Creatinin per kg. in mg.	Creatinin coefficient (Shaffer).
A.	4.95	20.65	4.2	1.56
B.	3.12	5.65	1.8	0.67
C.	3.72	6.48	1.7	0.65
D.	4.2	17.35	4.2	1.53
E.	4.3	8.5	2.0	0.73

In all these cases[1] the urine of the first 48 hours was collected under the necessary precautions. The reaction of the urine was always acid to litmus. The urine did not reduce Fehling's solution, but contained more or less albumin. The amounts of urine and the specific gravity were as follows:

	Urine of 48 hours in cc.	Specific gravity.
A	193	1005
B	27	1010
C	59	1003
D	58	1010
E	23	1008

The following short abstracts from the histories may illustrate the general condition of the infants:

Case A.—C., white (obstetrical No. 3831), weighed at birth 5190 gm. and on the third day its weight fell to 4720 gm., the lowest point reached. On the thirteenth day it surpassed its birth-weight, being discharged on the eighteenth weighing 5350 gm. Its length at birth was 55 cm.; it was born of a multipara. Its temperature and bowel movements were normal. It received the breast every three hours and from the second day some additional feeding of cow's milk.

Case B.—B., colored (obstetrical No. 3853), weighed at birth 3270, on the second day 3125 and on third 3070 gm. From this lowest weight it surpassed the birth-weight on the seventh and left on the fourteenth day, with a weight of 3600 gm. Its length at birth was 51.5 cm.; and it was a first-born child. Its temperature varied on the first day between 96.2° and 98.6° F.; on the second between 96.2° and 98.6° F. From the fourth to the seventh day the temperature did not reach 96° F. On the fourth day a jaundice was noted. The bowel movements were normal. The baby received the breast at three-hour intervals.

Case C.—C., colored (obstetrical No. 3912), weighed at birth 3850, on the second day 3710 and on third 3590 gm. From this lowest level the birth-weight was nearly regained on the fifth day. The baby left on the fourteenth day with a weight of 4145 gm. Its length at birth was 49.5 cm.; it was born of a multipara. On the fourth day the temperature became normal and remained so; previously it had been subnormal. The bowel movements were normal. The baby was put to the breast every three hours.

[1] The difficulty of collecting the urine of infants quantitatively without having a trustworthy person to watch its collection, made it impossible to examine a larger number of cases. That we were able to obtain the urine of our cases, we owe to the courtesy of Miss C. Turford, head nurse of the obstetrical ward of the Johns Hopkins Hospital, and we wish to acknowledge our indebtedness to her kindness. Our thanks are due, to not a small degree, to the interest and help which Dr. H. J. Storrs, resident obstetrician at the Johns Hopkins Hospital, has extended to us.

CASE D.—J., white (obstetrical No. 3943), weighed at birth 4350, on the second day 4180, and on the third 4020 gm. The lowest level of 4010 gm. was reached on the fourth day. The baby left on the fourteenth day without having regained its birth-weight. The weight was then 4300 gm. Its length at birth was 54 cm.; it was born of a multipara. The temperature reached normal on the fourth day. The bowel movements were normal. It received the breast every three hours.

CASE E.—M., white (obstetrical No. 3943), weighed at birth 4430, on the second day 4340, and on the third 4220 gm. The lowest level was reached on fourth day with 4200 gm. and it left on the fourteenth weighing 4409 gm., not having regained the birth-weight. Its length at birth was 56 cm.; it was born of a multipara. The temperature was normal, and also the bowel movements. The breast was given every three hours.

For several reasons some of the creatinin determinations are not absolutely exact. It is necessary to remove the albumin and with a few portions of urine this was accomplished by treating the urine with basic lead acetate. The lead is removed in the usual manner with H_2S; the filtrate from the lead precipitate is evaporated to dryness, and the residue is taken up in water and then filtered. Preliminary experiments showed that a small loss of creatinin occurs in this manner.

In order to avoid this loss it was deemed advisable to proceed as follows, boil the urine with a few drops of acetic acid and according to the specific gravity of the urine the filtrate was either used directly or after concentration. Care must be taken that the readings with the Duboscq calorimeter fall within the prescribed limits (1). For instance in Case A the reading for the part of urine treated with lead acetate was 4.35 and for the part boiled with acetic acid filtered and evaporated it was 4, while it should fall between 5 and 13. Under such circumstances the creatinin determination will result in figures which are too low.

The same example shows the loss due to the treatment with lead acetate. The total amount of creatinin was 41.3 mg. when the urine was not treated with lead acetate, and when this salt had been added it was 38 mg.

A further source of error in the lead-acetate method may be introduced in the evaporation of the voluminous filtrate from the lead precipitate. This is naturally rather strongly acid and the long heating may convert some creatin, if any is present, to creatinin, Mellanby (2) and Rothmann (3). In our preliminary experiments this source of error did not occur because the urine used did not contain any creatin. Former experiments have shown that under usual conditions the evaporation of the natural acid urine did not give rise to this error. In all cases determinations were made with urine not treated with lead acetate and in Cases D and E without evaporating the urine.

Comparing the results of Tables I and II it seems that the creatinin excretion during the first two days of life is smaller than later on. Naturally a larger series of cases will have to be examined before this can be accepted as a general rule and it will be necessary to follow the excretion in the same individual for some time.

The question arises whether the creatinin excreted by the infant during the first days could derive its origin from the amniotic fluid or the mother's blood. We were able to examine the amniotic fluid in two cases. In a case of hydramnion 1160 cc., and in a normal case 585 cc. of amniotic fluid were collected; its reaction was neutral to litmus.

After treatment with basic lead acetate the amount of creatinin in the 1160 cc. of amniotic fluid was found to be 16.8 mg. and when determined with the filtrate after boiling with acetic acid 17.9 mg. The total amount of creatinin in the 585 cc. of fluid was found to be 9.3 mg., after treatment with lead acetate. It is of interest to note that in these two cases the amount of creatinin in milligrams per 100 cc. fluid was nearly the same, in the case of the hydramnion 1.5 and in the other 1.6 mg. The possibility has to be considered, that the creatinin enters the amniotic fluid as a urinary constituent of the fœtus. But the preponderance of opinion seems to favor the view that the amniotic fluid is derived from the maternal blood serum, which has been altered by some specific activity on the part of the amniotic epithelium, Goenner (4), Williams (5). Whether or not the creatinin content of the amniotic fluid can be used as an indication that a urinary excretion of the fœtus enters into its composition, is a question which we will have to leave open.

The urine of Case D was collected again on the fifth and sixth days, and it contained 18.3 mg. creatinin for twenty-four hours, with 4.4 mg. per kilogram and a creatinin coefficient of 1.65; that is, the values obtained differed only very slightly from those of the first forty-eight hours. This result makes it rather improbable that the creatinin excreted during the first forty-eight hours should derive its origin to any extent from the amniotic fluid or the mother's blood.

With regard to the creatin, the urine of Case E only could be examined and none was found. In two cases previously reported a small amount of creatin was found and in the second case it was found in each of the six periods of forty-eight hours, only being absent or not present in determinable amounts during a period of water diet.

To complete the data collected thus far concerning the excretion of creatinin in infants the further results of Funaro (6) may be cited first in the following table:

TABLE III.

No.	Age.	Weight in gm.	Diagnosis.	Diet.	Total (24 hrs.) creatinin in mg.	Creatinin per kg. in mg.	Creatinin coefficient (Shaffer).
1.	2 mos.	3000	Lues cong.	Hum. milk, buttermilk.	21.0	7.0	2.6
2.	3⅓ "	2500	Lues.	Hum. milk.	17.25	6.9	2.6
3.	4½ "	4000	Enteritis.	Hum. milk, buttermilk.	24.7	6.17	2.3
4.	5 "	3900	Enteritis.	Buttermilk.	24.5	6.28	2.3
5.	7 "	4775	Chronic disorder of nutrition.	Buttermilk.	52.0	10.91	4.0
6.	9 "	4000	Atrophy.	Buttermilk.	22.5?	(5.5) 4.62??	2.1
7.	9 "	6500	Encephalitis.	⅔ milk.	24.0	3.7	1.4
8.	9 "	8300	Eczema.	⅔ milk.	60.0	7.23	2.7
9.	10 "	7200	Chronic disorder of nutrition.	Buttermilk.	37.95	5.27	2.0

Besides Funaro determined in three of his cases the creatinin output on different days and with different diets

		Diet.	Creatinin per 24 hrs. in mg.	
Table III.	8	⅔ milk.	60.0	Funaro.
		⅔ milk.	55.0	
		⅓ milk.	77.0	
		⅓ milk with casein and fat.	67.2	
		⅓ milk.	60.0	
Table III.	6	Buttermilk.	75.0	
		⅔ milk.	65.0	
		Buttermilk.	63.0	
Table I.	12	⅓ milk.	42.0	
		⅓ milk sugar.	40.0	
		⅓ milk with casein butter.	36.0	
		Casein and fat.	38.0	
Table I.	6	Human milk.	38.5	Amberg and Morril (7).
		Do.	29.6 (?)	
		Do.	37.6	
		Modified human milk.	33.2	
		Do.	29.9	
		Do.	31.4	

With regard to the creatinin nitrogen expressed in per cent of the total nitrogen of the infant's urine the following data are at our disposal. In the five cases reported by Amberg and Morril (8) concerning normal breast-fed infants between the ages of 7 and 13 days the creatinin nitrogen participated in the total nitrogen with 2.56, 3.6, 3.61, 2.98, and 2.72%. While the creatinin excretion per kilogram in the infant falls below that of the adult, the creatinin nitrogen participates in the total nitrogen output in the urine to the same extent as in the adult on a diet rich in protein.

In the other case reported by the same authors concerning a normal breast-fed infant about one month old, these figures were for the first three periods 2.2, 2.0, 2.4% and when the diet was modified to approach an "Erhaltungsdiät" they rose to 3.0, 4.3 and 4.5%. It will be of advantage to inquire whether the data thus far collected about the excretion of creatinin in the infant permit us to draw some conclusions particularly with reference to their possible clinical applications. This question leads us by necessity to a short consideration of the theories which have been advanced about the significance of the creatinin excretion. Folin (9) sees in the creatinin excretion an indicator of the endogenous nitrogen metabolism and makes it depedent on the active mass of protoplasm. At the same time he claims an entire independence between the creatin and creatinin in the economy of the organism. Shaffer (10) modified this view. The creatinin excretion is an index of some special process of normal metabolism taking place largely, if not wholly, in the muscles. Upon the intensity of this process appears to depend the muscular efficiency of the individual. He introduces his creatinin coefficient,[2] which expresses the amount of creatinin nitrogen in milligrams per kilo of body-weight in twenty-four hours. In strictly normal adults this creatinin coefficient varies between 7 and 11 mg. In pathological subjects this coefficient is usually low. The excretion of creatin on a diet free from it is referable to a rapid loss of muscle protein. Mellanby (l. c.) claims that the liver produces creatinin which in the developing muscle is transformed to creatin, and only makes its appearance in the urine when the muscle is saturated with creatin. With a loss of liver activity in pathological cases he found a diminution of the creatinin excretion. The muscles play a small part in the formation of creatinin. Folin's view concerning the entire independence of creatin and creatinin in the economy has lost in probability particularly through the experiments of Gottlieb and Stangassinger (11) (confirmed by van Hoogenhuyze and Verploegh (12) and Rothmann (3)). These authors demonstrated the presence of ferments in tissues capable of transforming creatin to creatinin, besides others capable of destroying either one. Van Hoogenhuyze and Verploegh assume that creatin is formed in different organs as a product of metabolism, and particularly in the liver. The circulating blood removes the creatin from the organs. A great part is further oxidized, another part is converted to creatinin. The same authors consider their results as a further confirmation of the hypothesis of Folin that a stimulation of the life process of the cells of the body expresses itself in an increased creatinin excretion. The small amounts of creatinin excreted a short time after birth are said not to contradict this view.

It is not proved that during this period of life the organism should not utilize to a much greater extent than later the chemical energy of the creatin formed in the body. In the infant the building up of new cells exceeds by far their destruction and therefore it is not improbable that an intermediary metabolic product like creatin is much better utilized than in the adult.

With regard to the theory advanced by Mellanby (l. c.) we should like to point out an interesting fact. Mellanby deduced from his experiments on the chick that the creatin content of the muscle is dependent on the growth of the liver. At a time when this organ enlarges rapidly the creatin content of the muscles increases rapidly, although the muscular growth almost stops. Creatinin is not excreted by chickens until about a week after hatching, *i. e.*, not until the muscles are saturated with creatin. All the data at our command show that the creatinin excretion of infants is certainly much smaller than in the adult. At the same time the liver is certainly larger in proportion to the body-weight during infancy than in adult life; the proportions given by Harley (13) (see Czerny and Keller) are in the new-born 1:18, in infants 1:20 and in the adult 1:50. The muscles of the new-born infant on the other side constitute only 23% of the body-weight, while those of an adult constitute 43% of the total weight (11). According to Mellanby's hypothesis one would

[2] In the tables we have given the creatinin coefficient of Shaffer. This coefficient is readily determined by multiplying the amount of creatinin excreted per kilogram by 0.372. Since this factor is a constant the coefficient varies directly with the amount of creatinin per kilogram and therefore it does not seem to offer any particular advantages over the calculation of the creatinin in milligrams per kilogram in twenty-four hours. It may be better to reserve the term creatinin coefficient to express the creatinin nitrogen in per cent of the total nitrogen similarly to the way in which the term ammonia coefficient is used.

predict *a priori* that this relatively large liver would rapidly produce sufficient creatinin to quickly saturate the small amount of muscle tissue with creatin and that subsequently there would be a relatively greater excretion of creatinin in the urine of the infant. This, however, is certainly not the case.

The paper of Mendel (15) on the physiological significance of creatin and creatinin furnishes a critical review of the hypotheses advanced by the different authors. From this exposé we learn that this problem is of a rather complicated nature and needs further elucidation. Therefore we will abstain from entering into a more detailed discussion, particularly with regard to the results thus far obtained in pathological cases, heeding the author's warning, "Clinical investigators seem to forget that in these experiments of nature one rarely deals with simple conditions where a single organ, such as liver or muscle, is independently involved."

Nevertheless, while the single steps by which the end result is reached need further elucidation, it seems to us that a few conclusions may be reached from the end result. Thus the work of Folin and others (van Hoogenhuyze and Verploegh, Klercker (16)) seems to establish rather firmly that the creatinin excretion may really serve as an indicator of the endogenous nitrogen metabolism; and furthermore, at present at least, the work of Shaffer (l. c.) makes the idea of a close connection between some process in the muscles and the creatinin excretion rather attractive. The relatively small creatinin output in the normal infant certainly could find its explanation to a large extent on this latter assumption as has been pointed out before. And the result obtained from the study of the nitrogen partition in the urine of baby Davis (Amberg and Morril) could again be explained on Folin's and particularly Shaffer's theory. In this case the creatinin nitrogen formed on an average 2.2% of the total nitrogen on regular breast feeding. On a diet consisting of human milk modified in such a manner that it approached an "Erhaltungsdiät" the creatinin nitrogen formed on an average 3.9% of the total nitrogen, rising to 4.5% on the lowest protein diet.

This relative increase of the creatinin nitrogen was not as high as that observed by Folin on his diet poor in protein. According to the theory of Shaffer this result should have been expected, since the muscles of the infant form a smaller percentage of the body-weight than in the adult. Under such conditions, therefore, it could hardly be expected that the products of the muscle metabolism participate to the same extent in the products of the endogenous metabolism as in the adult. We have referred repeatedly to the relatively small amount of creatinin per kilogram excreted by the normal infant, when compared to that excreted by the adult. Amberg and Morril were the first to show this condition on the basis of quantitative determinations with the 24-hour urine. These results were confirmed by Funaro, who, however, forgot to mention that two of the three conclusions he advances had been reached by these authors. His third conclusion based on the cases given above is that the individual variations under normal and pathological conditions are not very great and that also when the diet of the individual is changed. It must be conceded, however, that the variations observed in different individuals are not very inconsiderable, but neither an influence of the age nor of the diet nor of the condition of the infant can be made out definitely from the data given. Since the diet of the infant may be considered as practically free from creatin or creatinin, an influence on the creatinin excretion could not well be expected.

An influence of the age must exist due to the gradual changes of the organism to the adult state, but it may be easily understood that this influence will show itself strikingly only when longer periods of time are taken in consideration. Rather remarkable is the fact that the condition of the infant, which may be judged to a certain extent by the weight, should not have shown a more definite influence. Number 6 of Table III, a case of atrophy with a weight of 4 kg. at an age of 9 months, excreted 4.62 mg. creatinin per kilogram and had a creatinin coefficient of 2.1 mg. A mistake must have occurred, since according to the figures given by Funaro the creatinin excreted in milligrams per kilogram should read 5.5. A further discrepancy is noted; it seems that in the same case the creatinin excretion was followed on different days on different diets and its amount is given as 75, 65 and 63 mg. in twenty-four hours, while in the table it is given as 22.5 mg. These mistakes are the more unfortunate as a study of just such cases would be of particular interest. For it has been shown that a number of ferment activities of different organs has suffered severely in the atrophic infants.

The results of the investigations on infants to which we have alluded in this paper are not very numerous, and a rather limited number of pathological cases have been recorded and the information concerning these pathological cases is rather scanty. But it does not appear very promising that the creatinin determination should furnish us much additional clinical information of greater value. The possibility cannot be denied that in cases of malignant tumors the creatinin determinations may be of some value; and a further study, particularly of severer disorders of nutrition, is very desirable, and it is not precluded that the creatinin and creatin excretion may be of some prognostic value in such cases.

The results obtained in the first forty-eight hours are certainly rather remarkable, giving the lowest figures for the creatinin excretion as yet observed in infants. At present we must content ourselves with stating our results, leaving any effort at explanation to a larger number of observations and particularly to observations following the creatinin and creatin excretion for some time in the same individual.

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A METHOD OF SPLINTING SKIN GRAFTS.

By John Staige Davis, M. D.,
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Every surgeon has his own pet method of dressing skin grafts, and fairly good results are obtained when the grafted wound is dressed with gauze moistened with normal salt, or borax solution, 1:40; with overlapping strips of rubber protective; with dry gauze or powders; with silver foil; or when it is simply left exposed to the air without any dressing. However, there are many partial takes and failures for the reason that the grafts are not properly splinted after they are applied, and in consequence slip down with the dressings, or are floated off by blood or serum collecting beneath them. In order to overcome this difficulty it is necessary to reinforce the grafts with some material which has enough body to act as a splint, and at the same time is not too rigid to shape itself readily to any desired location. It is also very important that it should not adhere to the grafts and granulations, or cause too much pressure, and that there be free escape of any secretions into the dressings.

After experimenting with various materials, I tried a coarse meshed net, such as is used for curtains. It is made of loosely woven flat bars of cotton thread, surrounding openings about 1 cm. in diameter. It is necessary to have the openings approximately this size as smaller ones often become clogged. This proved too flimsy, and also became adherent to the grafts. So in order to increase the body of the fabric, after washing out the sizing and drying, I soaked the material in a rubber solution made up of pure gutta-percha, from 15 to 30 parts (depending on the stiffness of material required), and chloroform 150 parts, and found that after the chloroform had evaporated and the material was dry there was enough stiffness to give a very satisfactory splinting material. When prepared the net should be of a light greyish-brown color throughout.

The Sterilization Before Application is as Follows.—Cut in pieces as large as may be desired and separate them with one or two thicknesses of gauze. Place in a sterile jar, and fill it with 1:1000 bichloride of mercury solution. Change this solution three times with twelve-hour intervals, and finally allow the mesh to remain permanently in 1:1000 bichloride solution. It can be kept for a considerable time in this way (I have used it after keeping it nine months in the bichloride solution), although it is better to make up small quantities and often. The dry permeated material will keep indefinitely. No hot solutions must come in contact with the mesh during the sterilization or application.

Technic.—After the grafts are in place the mesh is taken out of the bichloride solution and thoroughly rinsed with salt solution, then dried with a sterile towel. A piece is cut large enough to allow a margin around the grafted area of from 5 to 10 cm. Then the material is applied and pressed snugly down on the grafted area and surrounding skin or granulations. Should the conformation of the part or wound not permit the mesh to be evenly applied, a few cuts with scissors will allow an infolding and accurate fitting, which is necessary in order that the splinting may be successful. The overlapping edges may be secured to the skin by strips of adhesive plaster when necessary. After the net is in position the dressing selected is applied, and the whole secured by a bandage.

Where the overlapping material rests on granulation tissue, it will be found that it can be lifted up at any time without causing pain or bleeding, as the granulations do not adhere to or grow into the bars of the impregnated material.

With this mesh in place the grafts can be observed from time to time with little or no danger of displacing them. The first dressing is usually made 36 to 72 hours after operation, and if the gauze next to the mesh has dried out, it must be thoroughly soaked with salt solution, and then carefully lifted with an instrument, while with a pledget of gauze the net is held down, as the dressing is raised from it, in order to guard against any displacement. Then the wound is irrigated with salt solution, and any secretions wiped away. The mesh is left in place from 4 to 10 days, and then can be removed without any difficulty.

Any type of dressing may be used over this material—silver foil, wet or dry gauze, etc., and I have found it particularly desirable in those cases where the grafted area was exposed to the air.

Conclusions.—The use of such a material permeated with

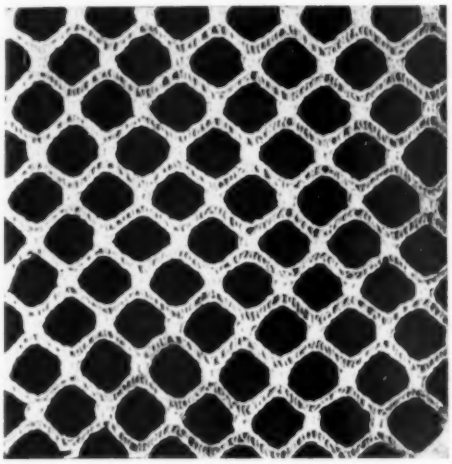

FIG. 1.—The actual size of the openings of the mesh.

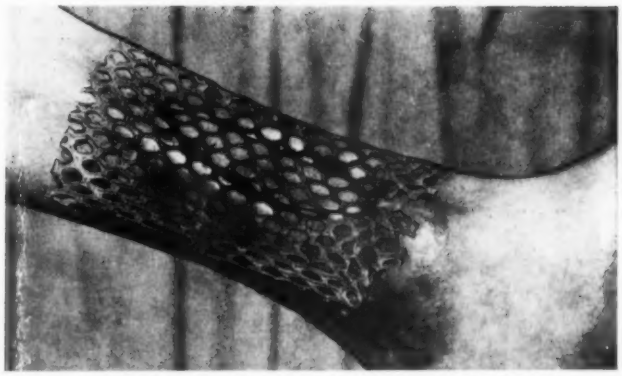

FIG. 2.—The material splinting a whole thickness graft on ulcer following osteomyelitis of tibia. Note cuts to allow accurate fitting. Photograph taken four days after application of mesh.

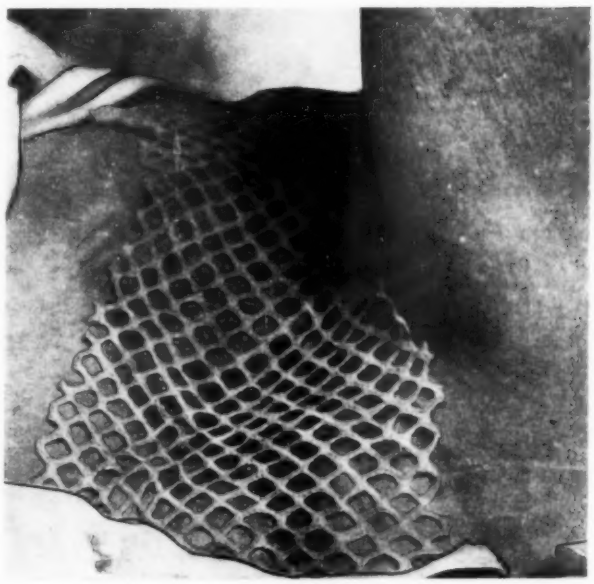

FIG. 3.—Illustrates the close fitting of the mesh over a Thiersch graft on deep breast wound following excision of carcinoma on a very fat woman.

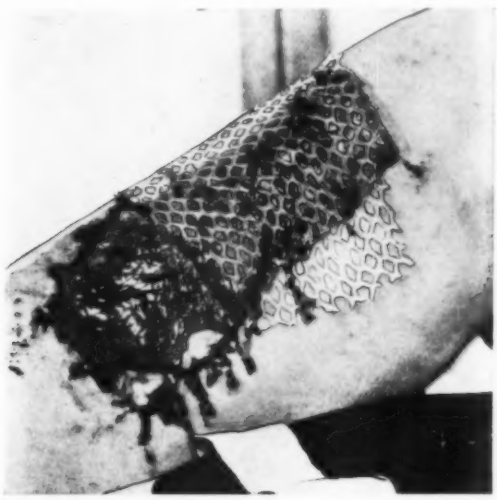

FIG. 5.—Mesh over partial whole thickness graft on ulcer of thigh. Photograph taken four days after application.

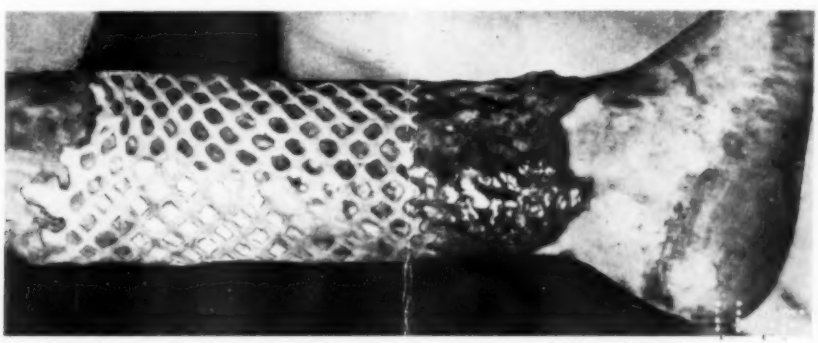

FIG. 4.—The mesh over whole thickness graft on burn, with the overlapping edges resting on granulation tissue. Photograph taken seven days after application

rubber is advantageous in that it splints the grafts without too much pressure, and is easy to apply and secure in place. It does not adhere to the grafts or granulations. It allows the free escape of any secretions which may form, and thus prevents maceration. Any sort of dressing may be placed over it. The progress of the healing may be observed at any time without danger of displacing the grafts. Should blisters form and serum or blood collect beneath the grafts, it can be removed at once.

I have used this open-mesh material over Thiersch and whole thickness grafts, on nearly every part of the body, and have found its use a distinct advantage.

NOTE.—This paper appeared in the Annals of Surgery, March, 1909.

A PRACTICAL METHOD OF IMITATING THE NORMAL AND ABNORMAL HEART SOUNDS FOR TEACHING.[1]

By CHARLES W. LARNED, M.D.,
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Dissatisfaction with the methods commonly employed in teaching the heart sounds, normal as well as abnormal, led me some years ago into the field of experimentation, my object being to produce, outside the body, tones which when conveyed to the ear would represent with a reasonable degree of exactness, at least in force and rhythm, the sounds heard through the stethescope placed upon the patient's chest.

Of the several methods studied I shall mention only four and describe in detail only one. In all instances the sounds are produced by taps, strokes and what I shall term tap-strokes and stroke-taps upon the conductor employed.

Method I. The left forearm is used as the conductor, the left hand being closely applied over the left ear, the taps and strokes being made upon the elbow.

Method II. The same, except that the taps and strokes are made not upon the elbow but upon the dorsum of the hand.

Method III. The stethescope is used as the conductor, the palm of the left hand being placed over the bell and the taps and strokes made upon the dorsum of the hand.

Method IV. The stethescope is used as the conductor and is applied to the right side of the bared chest, the taps and strokes being made upon the chest at a distance of some 2 to 4 inches from the bell. Distant and faint sounds can be produced by tapping over the pectoral muscles or in the intercostal spaces; loud and accentuated sounds by tapping over the ribs. The application of talcum powder to the surface stroked will soften the murmurs.

I shall now discuss briefly the merits and demerits of these several methods.

Methods I and II.—For over three years in my teaching I have employed the forearm as a conductor and I believe for normal heart sounds, for reduplications, and for normal and abnormal rhythm, this method gives the most realistic tones. A few weeks ago while demonstrating this method to my colleague, Dr. Henry Lee Smith, he suggested that the taps and strokes might be made equally well upon the dorsum of the left hand, and repeated trials have shown that certain abnormalities could be better imitated by following out this idea.

Methods III and IV.—Method III was given a trial but was soon discarded in view of the fact that the sounds conveyed are far too harsh. Method IV satisfies all reasonable requirements and I think is worthy of general employment. It has several advantages over the other methods, namely; the sounds produced are more realistic, the students become more rapidly familiar with the stethescope, and several students can be instructed at the same time. The great disadvantage in this method is that we must have a patient at all times for demonstration. I shall, therefore, in my detailed description deal with Method II, which can be employed without a subject, besides which this is the method best suited to those not familiar with the manipulations for producing the taps and strokes.

POSITION I.

To perfect oneself in the use of this method one requires but little practice, the only really troublesome sound to imitate being the presystolic rumble of mitral stenosis and even here the majority will have but little difficulty, a pianist should have none.

When practising upon oneself there are several positions that may be taken, but the two which give the best approximation for the ear hand and the freest manipulations for the percussing hand are as follows:

Position I. Turn the face well to the right and with the

[1] This paper was read before The Johns Hopkins Hospital Medical Society, December 20, 1909.

left forearm well back of the head place the hand closely about the ear.

Position II. Turn the face well to the left, place the left hand to the left ear, the forearm of the percussing hand being placed back of the head. The two photographs illustrate the two positions, in both of which the left hand acts as the conductor, the right hand as the plexor.

Definitions of the terms tap, stroke, tap-stroke and stroke-tap are as follows:

By *tap* I mean gentle percussion with the index or middle finger without movement of the forearm and with little, if any, at the wrist, the position of the finger being the same as in ordinary percussion, namely, flexed so that the terminal phalanx strikes the ear hand perpendicularly (otherwise the note produced is more or less impure), the strength and length of the tap depending upon the strength and duration of the note desired.

By *stroke* I mean the gentle drawing of the tip of the finger over the dorsum of the ear hand, a short stroke for

Position II.

short murmurs, a long stroke for long murmurs, the intensity of the murmur being indicated by the force of the stroke.

By *tap-stroke* I mean the direct merging of a tap into a stroke without any interval of time. It is made by ending the tap with a stroke before lifting the finger.

By *stroke-tap* I mean the direct merging of a stroke into a tap without any interval of time. This can only be done by the use of two fingers, the tap occurring simultaneously with the elevation of the stroking finger.

In demonstrating the method to a student it is well to have him place the right hand over the right ear with the elbow as far to the left as possible, and the face to the right. The instructor standing behind and slightly to the right makes upon the dorsum of hand so placed the taps, strokes, etc., which the condition he wishes to demonstrate may call for. A third photograph illustrates this position.

To obtain the best results the following points should be observed: The skin upon the dorsum of the hand should be smooth and not chapped, as a rough skin roughens the murmurs. A plump hand gives better imitations than a bony hand.

Accentuated sounds are best made by tapping directly over one of the metacarpal bones or over a knuckle; distant sounds are imitated by tapping over the muscles between the thumb and index finger.

Below are given as an example of what can be accomplished, the taps, strokes, tap-strokes and stroke-taps necessary to bring out the normal and abnormal sounds in some of the heart lesions most frequently met with. In each instance a diagrammatic representation is obtained by using certain symbols which with their equivalents are given below:

⌒ = tap, — = stroke } The force is indicated by the shading, the duration by the length.

⌒— = tap-stroke, —⌒ = stroke-tap } The force and duration are indicated respectively by the shading and length of each component part.

Vertical lines indicate the beginning of systole; the lettering shows the fingers used in the manipulation.

Position III.

The normal heart sounds as heard at the apex.—A light but rather long tap for the first sound followed by a lighter and shorter tap for the second sound, care being taken to observe the proper interval between the first and second sound, and the second and first sound.

Mitral insufficiency as heard at the apex—the murmur following and not replacing the first sound.—A tap-stroke made with the index finger to indicate the first sound and murmur followed by a light tap with the middle finger to indicate the normal second sound.

Aortic insufficiency as heard at the base, the murmur replacing the second sound.—A light tap made with the index

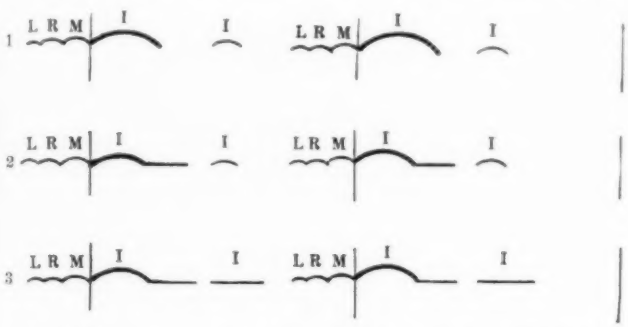
finger to indicate the first sound, followed by a stroke with the middle finger to indicate the diastolic murmur.

(1) *Mitral stenosis;* (2) *Mitral stenosis and insufficiency;* (3) *Mitral stenosis and insufficiency and aortic insufficiency; all as heard at the mitral area.*

(1) A rapid succession of four crescendo taps beginning with the little finger and ending with the index finger, the last tap being sufficiently strong to indicate the shock of the first sound, and immediately followed by a light tap to indicate the second sound.

(2) To bring out a systolic associated with the presystolic murmur convert the culminating tap of the presystolic rumble into a culminating tap-stroke, the stroke being lateral toward the little finger, light and short.

(3) To bring out a diastolic murmur associated with the presystolic or the presystolic and systolic make, instead of the



tap which represents the second sound, a short gentle stroke provided the murmur replaces the second sound, otherwise a tap-stroke, the stroke in either instance being lateral, toward the thumb, these two lateral strokes one for the systolic, the other for the diastolic murmur are best produced by a rocking motion at the wrist first to the ulnar then to the radial side.

Aortic stenosis as heard at the base.—A firm long scratching stroke made with the nail of the index finger to indicate the harsh systolic murmur, followed by a very gentle tap with the middle finger to indicate the relatively faint second sound.

Mitral and aortic insufficiency as heard at the apex, both sounds being replaced by murmurs.—A relatively long stroke with the index finger to indicate the systolic murmur, followed by a shorter and lighter stroke to indicate the diastolic murmur.

The pistol-shot sound heard over the arteries of the extremities in aortic insufficiency can be well imitated by a short sharp tap over the knuckle of the metacarpophalangeal joint of the middle or index finger.

Gallop rhythm, pendulum rhythm, reduplications, splitting of the first and second sounds and a variety of other conditions can all be well imitated.

In conclusion, I should like to state that I do not pretend to imply that by this method the sounds of the heart either in health or disease can be exactly reproduced, but what I do believe is, that the imitation of the rhythm, together with the time of occurrence of the normal as well as abnormal sounds, is sufficiently realistic to be most helpful to all teachers of physical diagnosis.

I wish before closing to express my thanks to Dr. Henry Lee Smith for having suggested the hand as a sound conductor, and also to give him credit for the imitation of the presystolic rumble which he so ingeniously worked out.

PROCEEDINGS OF SOCIETIES.

THE JOHNS HOPKINS MEDICAL SOCIETY.

October 18, 1909.

Dr. Frank R. Smith, the president, in the chair.

The first business of the meeting was the election of officers for the ensuing year, and Dr. Louis V. Hamman was duly elected president, and Dr. William L. Moss, secretary.

I. Demonstration of Tuberculides. DR. C. VON PIRQUET.

The case which I will demonstrate this evening, a boy three years of age, shows a symptom of cutaneous tuberculosis which is not yet much known in America. I am sure that it will be found here as frequently as it is in Europe since we learned to make the diagnosis of this insignificant looking efflorescence. Tuberculosis of the skin in its serious aspects has been known for a long time, and we make the distinction principally of lupus and scrofuloderma. Among tuberculous skin diseases, tuberculide is the one not yet much known. Tuberculides comprise the following:

1. Lichen scrofulosorum.
2. Erythema induratum (Bazin).
3. Folliclis.

This last form has been first described by Barthelemy in 1891. In later years only the knowledge of it has spread to the German clinics. F. Hamburger has made a special study of its clinical aspects, and Leiner proved the presence of tubercle bacilli in these efflorescences, whereas formerly Hallopeau had thought they were due to toxines only. In this case you see both kinds of folliclis; a *papular necrotic* one is to be seen on the elbow. You find a hard papula about ¼ of an inch in size, covered with white crusts. It is a chronic dermatosis,

but generally disappears after some months or years, leaving scars. In this case the mother has noticed it since July.

The other form is to be seen on several points of the chest and back. It consists of very small papules, about 1/10 of an inch in diameter, of a livid red color. The characteristic points are: a lack of any tendency to ulcerate, then a depression in the center, and finally the fact that the papula has a little gloss on it (F. Hamburger). The mother could not say when the efflorescences first appeared, but as they are not of a very fresh color we can tell that they have been there for several weeks; new ones have a more reddish color. This form is called *papulosquamous*, because there is often a delicate scale on top of it, and only after the scale is taken off, the depression is seen in the center. All the forms of tuberculides are a definite sign of tuberculosis. They are seen not infrequently in children of the first year. They have certainly the meaning of an active spreading of tuberculosis, as tubercle bacilli have been found in them. It depends probably only on the number of tubercle bacilli put into the circulation, and of the parts of the body where the bacilli are scattered, whether this spreading will be a fatal one or will be overwhelmed by the organism. So we find a dense appearance of tuberculides often combined with miliary tuberculosis. It is an interesting fact that this form of tuberculides has been found, in several instances, appearing two or three weeks after measles, and I think it is due to the lack of tuberculous allergy which we find during that illness. In this case measles have not preceded. We do not see now any symptoms of a miliary spreading of tuberculous germs, but we have several proofs of the existence of tuberculosis. The abdomen is large and contains probably free fluid; the child has a chronic discharge from both ears. You can see a marked new cutaneous tuberculin reaction. The test was also made July last, and already was positive then.[1]

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II. A Report on Some Cases of Purpura Hæmorrhagica following Benzol Poisoning. DR. L. SELLING.

This paper will be published in the BULLETIN.

DISCUSSION.

DR. T. R. BOGGS.—Dr. Selling's observations have much interest from a practical standpoint. In the main they correspond with those reported by Dr. Bunting in his experiments on rabbits using saponin and ricin as the toxic agents.

DR. W. S. THAYER.—It would be important to see if one could produce a chronic poisoning of the bone marrow with megaloblastic crises. I should like to ask Dr. Selling if he has observed any such crises in his experiments.

DR. SELLING (in reply to Dr. Thayer).—I have not. All the cells were of a normoblastic type.

DR. H. A. KELLY.—Has any precaution been taken at the factory to prevent any repetition of the poisoning?

DR. R. NORTON.—Mr. Tolliver, the superintendent of the factory, has lent every possible aid in this investigation as to the possible cause for these cases of poisoning. The factory is well lighted and well ventilated, and these accidents seem to have been unavoidable. A substitute is now being used for benzol in the sealing of the cans.

III. A Review of the Surgical Anæsthesia given in the Surgical Clinic of The Johns Hopkins Hospital. DR. W. D. GATCH.

This paper, together with further statistics, will be published in the BULLETIN.

DISCUSSION.

DR. W. S. THAYER.—It just happened that I was present in the Massachusetts General Hospital at the time of their annual anniversary of the introduction of ether as an anæsthetic by Dr. Morton. On Ether Day, as the anniversary is called, a paper is read concerning the use of ether and its applications to the medical science. President Eliot read a most interesting paper on Animal Experimentation, emphasizing what a great part ether played in the advance of medical science. I was also fortunate in seeing Dr. Morton's original apparatus for the administration of ether. It consisted of a large glass bulb supported on an adjustable stand, and having at its top two apertures, one to receive a long wooden mouthpiece, through which the patient was to draw the ether gas. The second aperture in the bulb was to receive a sponge saturated with the ether.

DR. H. A. KELLY.—I am glad Dr. Gatch has taken up this subject, and I hope he will include in his statistics those of the gynecological clinic of this hospital. To the best of my knowledge there has not been a single death attributable to the anæsthetic given in the gynecological service, or at my private sanitarium. We are not all so fortunate; only recently I witnessed the death of two patients on the operating tables of two very distinguished operators; in both cases the death could be attributed to the anæsthesia.

There is considerable danger in placing heavy, fat patients in the Trendelenburg position. The great weight of the intestines, and the heavy abdominal walls drag on the diaphragm and embarrass the heart's action. Twenty years ago I began using nitrous oxide as an anæsthetic, and I have never had cause to regret its use.

IV. On Paroxysmal Arteriospasm with Hypertension as an Accompaniment of the Gastric Crises of Tabes. DR. L. F. BARKER.

The patient shown was a white woman aged 49 years, a cigarmaker by occupation. Her complaint on admission to the hospital was severe pain in back and stomach with headache.

The family history was negative. The patient had been married twenty-four years; was the mother of six children, two of whom died in infancy. There is history of several miscarriages early in pregnancy.

Except for some vague rheumatic pains during the past ten years, she had suffered from no disease except the present illness.

Eight years ago the patient began to have pain in her back, indigestion, pain in the joints, especially shoulder and knees. Nausea occasionally accompanied the pain in the back. The attacks usually begin with the feeling of a lump in the throat, which the patient declares cannot be swallowed. Vomiting soon follows, and nothing can be retained upon her stomach. There is complaint of sensitiveness of the skin of the trunk. During the attacks there is no loss of consciousness, no disturbance of the sphincters; occasionally the patient has noticed palpitation of the heart. For the past year the attacks have averaged two per week, lasting from a few hours to three days. On examination, the patient was noted to be emaciated, the skin was sallow. Her eyes were rather prominent; there was a slight Von Graefe sign; the pupils were contracted and reacted but little to light and accommodation, but it was thought this might be due to the morphia, which she had been taking to relieve the pain. No glandular enlargement was found. The heart was slightly enlarged. The radials and temporals are tortuous and somewhat thickened. The lungs are negative, except for a moderate grade of emphysema.

An examination of the blood showed slight leucocytosis (13,800) and 92 per cent hæmoglobin. A differential count showed normal relations, except perhaps for slight increase of eosinophiles.

On October 13 the patient was observed by Dr. Barker, during a paroxysm of pain, and the following note was made: The face was very anxious, the lips cyanotic, eyes reddened and lachyrimose. One got the impression, at once, that the pain was that of organic disease. The radial pulse was 124, regular, but of high tension, feeling like a fine whipcord under the finger.

The blood pressure was measured at once and found to be about 190 mm. She was given an inhalation of amyl nitrite and the pressure fell at once to 90 mm. A short while after, however, the pressure again became high, rising to 200, and later to 210 mm. The knee jerks were over active, but the plantar reflexes were normal. The pupils did not respond to light. There was no tactile anæsthesia of the chest, but definite analgesia in large areas over the lower extremities. Nothing abnormal was made out on palpation of the abdomen. Examination of the urine revealed no albumin or casts.

A lumbar puncture was made by Dr. Kingsley and the spinal fluid found to be under pressure of 150-200 mm. The fluid was clear, colorless and contained 50 lymphocytes to the cubic millimeter. The serum-albumin test was positive.

Further sensory examination on October 15 showed both lower extremities almost wholly analgesic. There was also analgesia in each axilla. Touch was nowhere impaired and thermal sensation was not markedly involved. Wasserman's reaction was negative. The patient's pain was relieved by morphine. As soon as the vomiting stopped she was given small quantities of milk every two hours.

During the next five days she had only two attacks of nausea and vomiting. She began to have a good appetite and to feel better. Her blood pressure varied between 175 and 215 mm. until the 18th. On the 19th it was found to be only 120 mm., and since then, during the past ten days, the maximal pressure has varied between 110 and 125 mm.

In spite of the active knee jerks, the character of the vomiting, together with the sluggish pupils, and the analgesia of the legs made the diagnosis of tabes seem probable. This diagnosis receives support from (1) the extreme hypertension due to arterial spasm accompanying the attack, and (2) the results of the examination of the spinal fluid, namely, the increased lymphocyte count and the protein content.

We find attacks of severe abdominal pain similar to those present in this case in three distinct diseases:

I. Lead poisoning (abdominal colic).

II. Angina abdominis associated with arterio-sclerosis of the abdominal blood vessels.

III. Gastric crises of tabes.

In the preataxic stages of tabes we find a high blood pressure associated with the attack of abdominal pain and vomiting, while a low pressure is usually to be found in cases of tabes complaining of lancinating pains in the legs. The causation of the pain in the gastric crises of tabes has been much discussed. It seems probable that it is due to the irritation of the dorsal root fibers causing a reflex vaso-constriction in the splanchnic domain. According to Pal's theory the smaller vessels are constricted, and the high pressure on the arterial walls proximal to the area of constriction stretches the nerve plexuses in the vascular walls and causes the agonizing pain.

November 15, 1909.

Meeting of The Johns Hopkins Hospital Medical Society, Dr. Louis Hamman in the chair.

I. Exhibition of Surgical Cases. DR. JOHN W. CHURCHMAN.

CASE I. *Typhoid Osteomyelitis.*—Boy aged fourteen years, with history of an attack of typhoid fever fourteen months ago. The patient recovered from the fever but was left with a typhoid spondylitis which is still present.

A few weeks ago he returned to the hospital with a symmetrical swelling in the region of the tibial tubercle on each leg. These lumps are easily palpable and slightly tender.

"X-ray" plates show that the condition is undoubtedly one of osteomyelitis, situated in the epiphyses from which the tubercle develops.

The osteomyelitis was of typhoid origin, and of a low order of infection, as proved by a bacteriological examination. There are no signs at present of an acute stage of inflammation; no granulations were found. These symmetrical lesions are very rare, especially in the present site, for typhoid osteomyelitis is most frequently found in the ribs in adults. At operation the bone was exposed and in the area of the swelling the bone had not yet completely ossified. The left side showed a small defect, apparently the remains of an old cloaca.

It is interesting to recall that Roswell Park called attention to the fact that after typhoid fever there is often marked irritation of the osteogenetic centers, and great tendency for growth of the part especially in the epiphyses of the large bones.

Case II. *Anterior poliomyelitis.*—This patient, a young man aged 21 years, who came to the hospital 19 months ago with a marked contracture of the left lower leg, and functionless tibialis anticus and rectus femoris muscles. He has been under surgical treatment for the past year and a half, and has had several operations.

In brief, the first operation consisted in the transplantation of the sartorius muscle into the rectus femoris muscle. The leg was then put up in extension. At the second operation the external and internal hamstring muscles were divided, and the leg again put in extension. Attention was then directed toward the foot, which presented a marked degree of talipes valgus. A complete Phelps operation was performed and a transplantation of a slip from the extensor longus digitorum and the extensor longus hallucis into the paralyzed tibialis anticus done.

The combination of these operations gives a good functioning leg; the patient, who was entirely unable to use the leg in walking on admission, now walks quite well with a brace.

Dr. Churchman emphasized the fact that treatment should be begun from the very start to prevent the formation of contractures.

Operation is not indicated early, certainly not within the first year, as some of the muscles tend to recover their function. In all cases the functioning result is better than the anatomical result.

II. Exhibition of Case. Tuberculide. Dr. C. von Pirquet.

Dr. von Pirquet exhibited a most interesting case of tuberculide occurring in a child two years of age, who also showed a most marked positive cutaneous tuberculin reaction.

The so-called focus reaction first noted by Herd appears not only at the point of application of the tuberculin, but also in all of the areas of former injections. In this particular case about the points of injection, in addition to the "focus" reaction, several larger areas of the erythema multiforma type of cutaneous tuberculosis could be readily seen.

III. Discussion of Nephrotomy by the Silver Wire Method; with a Report of Two Cases. Dr. E. H. Richardson.

This paper will be printed in full in the Bulletin.

Discussion.

Dr. H. A. Kelly.—Mr. Max Brödel's work in the demonstration of the double vascular zones of the kidney has helped to simplify the operative technique of kidney surgery. We, as surgeons, are greatly indebted to him for his valuable contribution. I should like to hear from Mr. Brödel how he came to take up the investigation of this problem.

Mr. Max Brödel (in response to Dr. Kelly).—An artist should always have a clear conception of what he has to draw. He had, at times, found it necessary to do a good deal of original investigation before he could truthfully portray the object which he had to draw.

In this particular instance not finding sufficient knowledge on the distribution of the vessels in the kidney, he decided to work out the problem himself. Numerous celloidin injections were made, both of the arterial and venous systems. The results of these experiments Dr. Richardson has fully explained to you.

Mr. Brödel then, by means of blackboard sketches, showed just how he worked out the planes of arterial distribution. The determination of the direction of the incision in the nephrotomy operation was, curiously enough, suggested to him by attempting to pass a walking-stick through a thick privet bush.

Mr. Brödel noted how much more easily the cane could be passed through the bush if it was directed from the main trunks close to the ground and passed in an outward direction, than if one attempted to force the cane through the bush from without inward.

Believing the same would hold true in the passage of a blunt instrument through what he pleases to call the "renal forest of vessels," he decided that the proper direction should be from the pelvis outward to the cortex.

December 6, 1909.

Meeting of The Johns Hopkins Hospital Medical Society. Dr. Louis Hamman in the chair.

I. Conditions inducing Acapnia: a Phase of the Shock Problem. Dr. Yandell Henderson, Professor of Physiology, Yale University, New Haven, Conn.

"The regulation of the rate of alveolar ventilation in breathing depends under normal conditions exclusively on the CO_2 pressure in the respiratory center." Haldane and Priestly, in their experiments, subjected normal men to atmospheres varying widely in respect to the oxygen tension.

The respiratory movements of the subjects did not show any reaction to these variations, nor did the subjects themselves experience any alteration in their own condition. When the barometric pressure and the CO_2 content of the air breathed was altered, the respiration was found to vary in such perfect adjustment to these conditions that the tension of the CO_2 in the alveolar air was maintained nearly constant. The respiratory center is exquisitely sensitive to any rise in CO_2 pressure, and is equally sensitive to even a slight reduction in the tension of the CO_2 below the normal.

It has been known for years that vigorous artificial respiration is followed by a period of apnœa. This period of apnœa is due to the temporary lowering of the CO_2 tension of the arterial blood below the threshhold exciting value for the respiratory center, and Haldane and Priestly have found that in man this period of apnœa is not present when the air forcibly respired contains CO_2 at nearly the same tension as that of the alveolar air.

According to Miescher the period of apnœa may be said to

consist of two conditions, apnœa spuria and apnœa vera. The temporary inhibition of the respiratory center, through the stimulation of the afferent nerve endings of the vagi in the lungs, results in the former condition. Apnœa vera is caused by the over aeration of the center, by excessive pulmonary ventilation. The changes in the blood might consist in an over-oxygenation or in a reduction of its CO_2 content. The work of Haldane and Priestly, Miescher, Mosso and Fredericq have shown that when the CO_2 of the arterial blood is diminished apnœa vera results.

Crile has found in his numerous investigations on surgical shock that in 90 per cent of the subjects death was due to respiratory failure. "In many instances the heart was beating strongly and the blood pressure was fair, at the time respiration failed. The results of these experiments would go far to show that the diminution of CO_2 is a factor in surgical shock were it not for the fact that two other explanations of the regulation of respiration and causation of apnœa present themselves.

The first explanation is that respiration is essentially a reflex function, and that the center is the constant recipient of augmenting and inhibitory influences of afferent nerves; the other the hypothesis that surgical shock is but the exhaustion of the center by the stimulation of the afferent nerves. Recent investigations have tended to show that the excitement of the vasomotor center in asphyxia is caused rather by the excess of CO_2 in the blood than by the lack of oxygen.

The amount of CO_2 in the blood is diminished whenever the subject is under diminished barometric pressure, and this diminution is thought, by many observers, to be the cause for the disturbance of function present in the so-called mountain sickness. For this peculiar condition experienced by individuals when at great elevations, Mosso has suggested the term "acapnia" (from κάπνος, smoke, literally smokelessness). Many writers have commented upon the similarity of surgical shock and mountain sickness.

It is becoming a well-established fact that acapnia immediately produces tachycardia, and, if long continued, always results in shock. It is quite possible through breathing exercises for an individual to acquire ability to voluntarily increase or diminish the rate of pulmonary ventilation. For many centuries these breathing exercises have been practiced in India, with the belief that the subject can gain control over the heart rate and other functions. It is possible for the subject to render himself insensitive to pain or even unconscious. A condition of mental exaltation and hallucination can be produced.

Discussion.

Dr. W. H. Howell.—I wish to express my great appreciation of Dr. Henderson's paper. While many of his views do not coincide with the views of orthodox physiologists, the great importance of his paper in bringing to the front and emphasizing the subject of the relation of acapnia to shock will undoubtedly open up a broad field for fruitful experimentation.

I must confess that I knew that Dr. Henderson was to speak on this subject, but understood his paper was to be delivered at a meeting two weeks from to-night, and I would then have come prepared and fortified to discuss the paper in open meeting.

In the case cited by Dr. Henderson of a man dying from a "Fourth-of-July accident," in which death could not be attributed to hæmorrhage or shock, I am sceptical to believe that the patient died of acapnia pure and simple.

It has not been my experience in animal experiments to have the period of apnœa preceded by a period of hyperpnœa, and in the majority of the cases I have found the vasomotor center to hold out to the end, as has been the experience of Dr. Crile. I should like to defend the orthodox view of the automaticity of the respiratory center. So far as I am aware that center is automatic.

Dr. William H. Welch.—I remember reading, in one of the old works on surgery, of the custom of having a patient perform forced respiration for a few minutes just prior to a minor surgical operation, such as the opening of abscesses, etc. The belief was that this forced respiration produced a lessened sensibility to pain.

II. A Season's Experience with Prophylactic Vaccination against Typhoid.

Major F. F. Russell, U. S. A.

The adoption of antityphoid inoculation or vaccination under certain conditions appears to be warranted. The presence of epidemics so frequently seen in American cities and during the time of great international conflicts makes it desirable to adopt some method for protecting the uninfected. The utility of this protection against typhoid was readily demonstrated in the late campaigns of the British army in India and South Africa (Boer war). The occurrence of typhoid in those inoculated was one-half that among the uninoculated, and the inoculation reduced the mortality of the disease by one-half.

Following the injection of the vaccine a local reaction appears at the site of inoculation. This consists of a red, tender spot about the size of the palm of the hand, appearing six to eight hours after inoculation, and disappearing in forty-eight to seventy hours. Occasionally there has been some lymphangitis along the course of the lymphatics and slight enlargment of the axillary glands. No case of suppuration of the glands has been reported.

The constitutional symptoms are limited to a feeling of lassitude, headache and occasionally a mild diarrhœa.

The prevalent idea of the "negative phase" or period of increased susceptibility to the disease makes widespread inoculation impossible. The negative phase is quickly followed by a positive phase, the antibodies increasing rapidly. Twenty-four hours after the administration of small doses the positive phase can be demonstrated, and it is only when large doses are given that one obtains a prolonged negative phase. Wright advocates the giving of several moderate doses eight to fourteen days apart, rather than the giving of a single injection. The inoculation of these quanta induces an ample elaboration of anti-tropic substances (antibodies) without producing any severe constitutional reactions.

The bactericidal and agglutinating properties of the patient's serum are increased and a greater resistance to typhoid intoxication is established. Milder symptoms follow the injection of the second dose than after the first.

So far as it has been possible to ascertain, the protection lasts two or more years, although a few cases have been reported in which infection has taken place from three to six weeks after vaccination.

DISCUSSION.

DR. W. H. WELCH.—It is somewhat striking that the attitude of the people toward typhoid vaccination is different than that toward vaccination against smallpox. The fact that typhoid is not a very contagious disease may account in no small degree for the prevailing attitude. The comparison is hardly a fair one, since the vaccination against smallpox produces an enduring immunity, while in the case of typhoid the period of immunity is quite variable. Antibodies are produced, but as yet we have no index for determining just how much of an immunity we have obtained, other than exposure to the disease, at different periods after the inoculation.

The negative phase is practically a negligible factor, and is really not such a great obstacle. Results are much better, so far as question of mortality is concerned in the cases, where the patients have been vaccinated. A single dose does not give much immunity, but in cases where a second or even a third dose is given, quite a substantial immunity is obtained.

DR. W. W. FORD.—Castellani in his experiments advocates the giving of first, an injection of a culture of living typhoid organisms; this to be followed by an injection of the killed bacilli. In certain cases injections of mixed cultures of typhoid bacilli and bacilli of dysentery or typhoid and paratyphoid bacilli have proven efficacious.

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NOTES ON NEW BOOKS.

Surgical Diagnosis. By EDWARD MARTIN, M. D. Illustrated. Price $5.50. (*Philadelphia and New York: Lea & Febiger, 1909.*)

Dr. Martin has covered the ground of surgical diagnosis well in this volume of 740 pages, all of which is written by himself with the exception of brief chapters on Laboratory Diagnosis by Dr. Longcope, The Application of the X-rays by Dr. Pancoast, Gynecological Diagnosis by Dr. Auspach, Diagnosis of Nervous Affections by Dr. Weisenburg, and assistance in the chapter on the Eye from Dr. de Schweinitz. The author has considered all the more common surgical troubles critically, laying especial stress on the early clinical diagnosis, and as a text-book for students it will be received cordially by the profession.

A System of Operative Surgery. By Various Authors. Edited by F. F. BURGHARD, M. S., F. R. C. S., etc. In Four Volumes. Vol. II. Price $10.00 per volume. (*London: Henry Frowde and Hodder & Stoughton, 1909.*) *Oxford Medical Publications.*

The operations described in this volume are those for Tuberculous Affections of the Bones and Joints by Harold J. Stiles; for Hare-lip and Cleft-palate by Edmund Owen; for Cancer of the Lips and Face by G. Lenthal Cheatle; upon the Jaws and the Oesophagus by C. H. Fagge; upon the Tongue, Tonsils and Pharynx by H. T. Butlin; upon the Stomach by B. G. A. Moynihan; upon the Intestines by G. H. Makins; for Hernia by Arthur C. Barker; upon the Rectum and Anus by F. Swinford Edwards. The contributors have shown more judgment than is often seen in the amount of space devoted to their special chapters, and as a result, the volume is well proportioned. Each subject has been carefully and judiciously handled, and the better and more well-known operations fully described. There can be little doubt that much of the success of the volume is due to the editor's ability, and that his must be the credit for producing such a serviceable surgery. The work is abundantly illustrated, though many of the drawings are merely diagrammatic, and their quality is not as good as is to be found in many American text-books of a similar nature. However, they illustrate sufficiently well the different points brought out in the text. Otherwise the volume is an excellent piece of book-making.

Bier's Hyperemic Treatment. By WILLY MEYER, M. D., and PROF. DR. VICTOR SCHMIEDEN. Second Revised Edition, Enlarged. (*Philadelphia: W. B. Saunders Company, 1909.*)

This interesting subject is brought to the attention of the profession in an attractive and practical manner. The style of the

authors is easy and the many excellent photographs render the text readily understood. The authors are undoubtedly enthusiasts on the subject and have given much time to it. The success of Bier's hyperemic treatment depends upon a thorough knowledge and application of the proper technic. It is a treatment which demands a great deal of the surgeon's time and constant supervision by him and for this reason has doubtless proven unsatisfactory to many investigators. It has, however, a wide field of usefulness and every surgeon should be familiar with it. Although it is claimed that patients can be taught to apply the treatment themselves, this has been found unsatisfactory, except in a few cases. It seems to us it is essentially a treatment for a clinic or hospital where the patients can be under observation for a large part of each day. As a method for treating the various tubercular affections, while it is valuable, we do not think it will ever supplant the cast and brace treatment, for inexperienced hands these older methods will give excellent functional results, which is one of the advantages claimed for hyperemia. The average surgeon will never succeed in obtaining the results reported by Bier and the authors. Hyperemia, however, is well worth a permament place in our therapeutic armamentarium and the author's book should be read by all physicians who are desirous of keeping up with the progress of modern medicine. The statement by Meyer and Schmieden that hyperemia is not a panacea is certainly necessary, as otherwise one would be left under the impression that there was very little it did not do in combatting the various acute and chronic inflammatory diseases to which the human body is subject. HENRY W. KENNARD, M. D.

Introduction to Practical Chemistry for Medical, Dental and General Students. Specially adapted to meet the requirements of the Conjoint Boards Examination at the Royal College of Physicians and Surgeons, but suitable for general use in schools and for private students. By A. M. KELLAS, B. Sc., Ph. D., Lecturer on Chemistry at the Middlesex Hospital Medical School. (*London: Henry Frowde and Hodder & Stoughton, 1909.*)

This is one of the "Oxford Medical Publications"; like the majority of English text-books on chemistry it is written to prepare for a certain examination, hence is of little value for American conditions. Its contents are a few simple inorganic preparations, a brief treatment of qualitative analysis and a briefer treatment of quantitative analysis; the methods of qualitative separations are all old, and the subject is presented mainly in the old-time tabular form. E. RENOUF.

The Physician's Visiting List for 1910. Price $1.00. (*Philadelphia: P. Blakiston's Son & Co.*)

This visiting list, intended for the pocket of the busy practitioner, has succeeded where others have failed, for it is now in its 59th year of existence. It contains a calendar, a new table for calculating the period of gestation, table of signs, incompatability, poisoning, metric system, dose table, etc. Its appearance is neat.

Manual of Chemistry. A guide to lectures and laboratory work for beginners in chemistry. A text-book specially adapted for students of medicine, pharmacy and dentistry. By W. SIMON, Ph. D., M. D., Professor of Chemistry in the College of Physicians and Surgeons, in Baltimore, and DANIEL BASE, Ph. D., Professor of Chemistry in the Maryland College of Pharmacy. Ninth Edition, thoroughly revised. (*Philadelphia: Lea & Febiger, 1909.*)

The fact that this is the ninth edition of this comprehensive manual shows that it is so popular and well known that it needs little comment. It differs from all other manuals in the colored plates indicating the color of various precipitates of metallic and organic compounds. More important is the scope of the book, with introductory chapters on chemical physics, and the inclusion of inorganic, qualitative, quantitative and physiological chemistry in one volume. The volume contains 716 large pages, but to crowd the mass of information into this space requires a condensed style. The new edition, revised with the able co-operation of Professor Base, is a notable improvement on the previous editions because of the modern chemical theory introduced and the additions to the chapter on physiological chemistry.

E. RENOUF.

Organic and Functional Nervous Diseases. A Text-Book of Neurology. By M. ALLEN STARR, M. D., Ph. D., LL. D., Sc. D. Third Edition, Thoroughly Revised. Illustrated with 300 engravings in the text and 29 plates in colors and monochrome. (*New York and Philadelphia: Lea & Febiger, 1909.*)

The previous editions of this work have established its high place in the opinion of all who have had opportunity to use it and the present edition will, if possible, move it up a little higher in general estimation. Much additional matter has been added from the researches and experience of the author and from the literature of neurology. New chapters have been added on symmetrical gangrene, reflex neuroses, angio-neurotic œdema and trophic symptoms of mental disorders. Much new material has been added to the chapter on paresis, to the section on decompressive operations in diseases of the brain, and to the discussion on spasmodic neuroses. The mental symptoms of neurasthenia and psychasthenia are presented and under their treatment psychotherapy is admirably discussed. The space allotted to the functional nervous diseases has been doubled. All of these additions have added greatly to the value of the book and there is but little opportunity for criticism. What there is being more a matter of personal opinion than any real defect it does not seem proper to mention.

Dr. Starr has a very clear style of writing which makes his book delightful reading, and after looking up a certain point one is tempted to read on. To the student this is an extremely valuable characteristic which is unfortunately not always found in text-books prepared for medical students. The illustrations are very good and the general make-up of the book is excellent. We have nothing but praise to say for this edition which we hope will be even more successful than its predecessors. W. R. D.

Diseases of the Genito-Urinary Organs and the Kidney. By ROBERT HOLMES GREENE, A. M., M. D., and HARLOW BROOKS, M. D. Second Edition, Revised and Enlarged. Price $5.00 net. (*Philadelphia: W. B. Saunders Company, 1908.*)

This book is the conjoint product of a surgeon and a physician. Chapters on subjects not usually incorporated in books of this character have been introduced, such as as: "The Blood in Diseases of the Kidney.': "Ocular Manifestations of Renal Disease," and a chapter on "Bright's Disease" The portion devoted to methods of examination of patients and manner of taking histories is valuable to the student. In the chapter on "Acute Urethritis" the writers state as a result of their clinical experience that it is better to postpone local treatment until the acute stage has passed: this is usually the 4th to the 6th week after the onset of the disease. In view of the fact that a very large percentage of cases are cured by the institution of local treatment within a few weeks, this seems strange advice. Even in cases where a rapid cure is not effected we have never seen any harm result where treatment was rationally applied. The article on stricture of the urethra is very poor and contains many errors. For instance, they state that the majority of strictures occur in the membranous urethra. All other writers, as far as we are aware, are unanimous in their statements that strictures origina-

ting in this region are rare. Sir Henry Thompson searched the pathological museums of Europe and was unable to find one specimen with stricture in the posterior urethra. The above is only one of many statements with which we cannot agree. The portion devoted to diseases of the prostate could profitably be rewritten. We most strongly disagree with the authors that prostatic massage is best performed by rubbing around the prostate and avoiding all pressure over the gland. It is little wonder they do not seem very enthusiastic about the results to be obtained from prostatic massage. As a whole the book is distinctly below the average, particularly in the portions devoted to disease of the lower genito-urinary tract.

Studies in Rabies. Collected Writings of NATHANIEL GARLAND KEIRLE, A. M., M. D., etc. With an Introduction by WILLIAM H. WELCH and a Biographical Sketch by HARRY FRIEDENWALD. Testimonial Edition. (*Baltimore: The Lord Baltimore Press. 1909.*)

It is fitting that the papers of Dr. Keirle, who was the founder of the Pasteur Institute in Baltimore, and whose articles on rabies are of distinct value, should be collected in a volume for the benefit of the profession. Such a testimonial is a mark of esteem of the man, as well as of the worth of his work. The volume is an important contribution to American medicine in a branch to which little has been added by American doctors, whose opportunities to study it have not been limited. It is to be hoped that his example may stimulate others to pursue further investigations in this horrible disease, which frightens the public almost more than any other. Any more contributions in this country in regard to its control and treatment will be largely due to his initial studies.

A Text-Book of Genito-Urinary Diseases. By DR. LEOPOLD CASPER. Translated and edited, with additions, by CHARLES W. BONNEY, B. L., M. D. Second Edition, Revised and Enlarged. Price $5.00 net. (*Philadelphia: P. Blakiston's Son & Co., 1909.*)

The second edition of this well-known work has been changed only in minor details. More attention has been devoted to a discussion of acute and chronic urethritis.

For a volume of its scope the contents are complete and the subject matter well handled. It is a work well suited for students and both the author and translator deserve commendation.

Etudes Anatomo-Cliniques: Coeur Vaisseaux Poumons. Par DR. RAYMOND TRIPIER, Professor à la Faculté de Médecine de Lyon. 595 pages. (*Paris: G. Steinheil, Editeur, 1909.*)

The book is conceived in a spirit of protest against the tendency to separate sharply the studies of pathological anatomy and clinical medicine. Specialization is thought to have exceeded its just bounds, missing the fruits of an intimate correlation. Throughout these studies, anatomical considerations are closely interwoven with ingenious reasoning from clinical data. The material put before the reader, while not always convincing, is interesting. Most characteristic are the chapters on tuberculous endocarditis, arterio-sclerosis, and incipient pulmonary tuberculosis. To tuberculous endocarditis, the author gives a larger sphere than is usually conceded in similar works.

In the chapter on arterio-sclerosis, the author builds largely on his view that inflammation is really in large part a mechanical change consequent upon capillary obstruction. He dissents strongly from the views of Huchard. The vascular lesions in arterio-sclerosis are regarded not as the result of hypertension consequent upon renal changes, but secondary to changes in many organs due to the same cause that induced the renal lesion. Hypertension consequently is the result, not the cause, of arterio-sclerosis. Hypertension produces not degeneration, but hypertrophy of the heart, and hypertrophy alone is the usual finding in the *Brightique* hearts of young people, unless there is a history of previous infection such as might be sufficient to produce tissue changes in heart muscle.

The author's views on pathogenesis of pulmonary tuberculous lesions are redeemed by his radical but sound advice as to treatment. Tripier does not share in the prevalent but unjustified optimism as regards the prognosis of definitely recognizable pulmonary tuberculosis, and therefore urges treatment during the so-called pre-tuberculous stage. In this he touches upon a sore difference between those phthisotherapeutists who emphasize the frequent recovery of patients with well-established lesions, and those who have been distressed by the unfortunate outcome in some cases which have received treatment even before a diagnosis could safely be made. S. W.

Proceedings of the Third Annual Conference of the American Association of Medical Milk Commissions. 1909. Cincinnati, Ohio.

This association, now in its fourth year, deserves support and endorsement. Its members are striving actively in their efforts to secure pure milk supplies all over the country, and thus to help in diminishing the mortality and morbidity which results from dirty milk. It is questionable whether there is any other food consumed so dirty as milk ordinarily is. It should be one of the first duties of health boards to enforce pure milk supplies, and this association is doing good work towards this end.

Diseases and Surgery of the Genito-Urinary System. By FRANCIS S. WATSON, M. D., etc. Assisted by JOHN H. CUNNINGHAM, JR., M. D., etc. Illustrated. 2 vols.. Price $12. (*Philadelphia and New York: Lea & Febiger, 1908.*)

This work is in two volumes and is by far the most complete and thorough treatise on diseases of the genito-urinary tract published in English.

The first volume is devoted to the external genitals, prostate and bladder, while the second volume deals almost solely with the kidney. Tuberculosis of the genito-urinary tract is considered as a whole in a chapter at the end of the second volume. This seems a very rational method of handling the subject instead of considering it in a fragmentary manner under diseases of the prostate, again under testicle, bladder, etc. Various portions of the tract are so frequently involved at the same time that the student can only get a clear concept when the disease is considered as a unity.

An unusual amount of space has been devoted to the description in detail of operative procedures. The illustrations in the work are good and the photographs of specimens showing prostatic hypertrophy are splendid.

Possibly for use of the average student sufficient space has not been devoted to the minor subjects. The authors have evidently devoted time and study to the preparation of the volumes, furnishing each section with a good bibliography and have produced a work which is to be highly commended.

The work impresses one as being the product of men with a familiarity and thorough grasp of the subjects handled.

Biographic Clinics. Volume VI. Essays Concerning the Influence of Visual Function, Pathologic and Physiologic, upon the Health of Patients. By GEORGE M. GOULD, M. D. (*Philadelphia: P. Blakiston's Son & Co., 1909.*)

We regret to learn from the author in the first chapter that "This forelying sixth volume of Biographic Clinics will be the last I shall write. To the gospel I have given all the strength

and money I could spare and of both the expenditure has been great." Those who have read the previous volumes cannot help regretting that we are not to have similar pungent essays and trenchant statements as to the effect of errors of refraction upon the health of patients. Granting that like all other souls crying in the wilderness the author has at times seemed impatient of the slow progress of his evangel, no one can aver that he has not brought about great results. The attention of physicians and others has been directed to this potent cause of ill-health and it is now the custom to seek diligently for errors of refraction in the study of all cases of obscure disease. The injurious effect of uncorrected astigmatism upon bodily function is now generally acknowledged, and for the acceptance of this addition to our knowledge we are largely indebted to Dr. Gould. The zeal of the accomplished author sometimes betrays him into a vehemence of expression not wholly in harmony with an exposition of scientific truth. While this may add to the vigor of the book, it does not add to the permanent effectiveness of the appeal and is to be deplored. "The Case of Jonathan Swift" and "The Pessimist—Added Testimony in Wagner's Case" are valuable contributions to the life histories of well-known men and of equal interest to similar biographies of Carlyle, Elizabeth Barret Browning and others in previous volumes.

A Manual of Otology. By GORHAM BACON, A. M., M. D., Professor of Otology in the College of Physicians and Surgeons, Columbia University, New York. With an introductory chapter by Clarence J. Blake, M. D., Professor of Otology in the Harvard Medical School, Boston. New (5th) Edition, Thoroughly Revised. 12mo. 500 pages. 147 engravings and 12 plates. Cloth, $2.25 net. (*Philadelphia and New York: Lea & Febiger, 1909.*)

We have previously reviewed this excellent little manual and it is a pleasure to note that the author has so soon been called upon for another edition. The book fully covers the needs of general medical students and family practitioners of medicine and deserves the favor it has received in these fields. The introductory chapter, pointing out the importance of this special branch of medicine and the relationship of otology to general medicine should have the consideration of every physician.

The alterations in the new edition, bringing the whole work quite up to date, are a pleasing feature and Dr. Bacon is to be congratulated upon having presented the students with one of the most complete and compact manuals in the English language.

H. O. R.

The Medical Complications, Accidents and Sequels of Typhoid Fever and the Other Exanthemata. By H. A. HARE, M. D., B. Sc., Professor of Therapeutics in the Jefferson Medical College and Physician to the Jefferson College Hospital, Philadelphia, and E. J. BEARDSLEY, M. D., L. R. C. P., Philadelphia. With a special chapter on the Mental Disturbances Following Typhoid Fever, by F. X. DERCUM, M. D., Professor of Nervous Diseases in the Jefferson Medical College. Second Edition, Thoroughly Revised and much Enlarged. Illustrated. Price $3.25. (*Philadelphia and New York: Lea & Febiger, 1909.*)

The second edition of this book differs from the first in the addition of new chapters on the complications, etc., of variola, scarlet fever, measles, varicella and rubella; and an excellent chapter by Dr. Dercum on the mental disturbances following typhoid fever. The work is a useful one, but has not been prepared with sufficient care. For instance in the chapter on rubella, there is only one reference since 1900, and most of them are much earlier. The typesetting also in this chapter does not agree with that used in the other chapters. There is still one severer criticism to be made of the work as a whole. The bibliography throughout has been prepared in a slipshod manner, entirely wrong references are given—both as regards titles and dates—the latter being frequently omitted. There is no general system of abbreviations used, and the misprints in the titles of foreign journals are inexcusably bad; there are wrong spellings, accents, capitals and punctuation. The errors are numberless; the reviewer has not taken the trouble to look up each individual reference, for the mistakes are self-evident. Important references that should be given are entirely omitted. In a work of this nature, where it is all essential that the references should be correct, such a bibliography has no excuse, and injures the value of the book for all students. It is to be hoped that a third edition will be "thoroughly revised."

A Text-Book of Special Pathology for the Use of Students and Practitioners. By J. MARTIN BEATTIE, M. D., Professor of Pathology and Bacteriology, University of Sheffield, etc., and W. E. CARNEGIE DICKSON, M. D., Lecturer in Pathology and Bacteriology, and Senior Assistant to the Professor of Pathology in the University of Edinburgh, etc. Price $5.00. (*Philadelphia: P. Blakiston's Son & Co., 1909.*)

The authors say in their preface, "If any excuse were necessary for adding another to the many text-books of pathology, it would be found in the fact that the present volume is based on the teaching of the Edinburgh School." There can be no doubt concerning the influence which this school has had in moulding pathological opinion and inasmuch as this book deals with pathological problems as they are conceived by that school, led at present by Dr. Greenfield, it will be read with interest by the pathologist and the student.

The book corresponds in its scope to the numerous special pathologies intended for the student and practitioner already on the market. Its arrangement presents nothing radically opposed to what we are accustomed to find in works of this nature. It is written in an exceptionally clear and logical manner so that the reader readily grasps the underlying thought and best of all it is elaborately illustrated by splendid photographs of typical morbid conditions. These deserve special mention since they are not the old hackneyed pictures one is accustomed to see in books of this nature. The photomicrographs are exceptionally clear and illustrative.

The relation of the gross and microscopic pictures of morbid conditions, probably one of the most difficult obstacles for the student to conquer, and also an extremely difficult subject to be explained by descriptive writing is only briefly dealt with.

At the beginning of many chapters a brief résumé of the normal anatomy and histology is found which will be of great assistance to the student. He finds, for instance, a clear and concise description of the normal structural arrangement of the complicated system of renal tubules, vessels, etc., before the pathology of this organ is considered and again before the discussion of malformation of the heart and great vessels is entered upon one finds a brief description of their embryonic development which is so necessary for the proper understanding of their malformations.

The discussion of some rather difficult problems is particularly clear. Among these it is of interest to note that the authors consider Hodgkin's disease a distinct clinical and pathological entity entirely independent of the tubercle bacillus, and to be rather of the nature of an infectious granuloma than a new growth. They describe the condition practically in the manner of Miss Reed, though they do not quote this author and yet quote in detail the later work of Longcope.

Concerning the nature of the pathological change in the liver cells consequent upon chronic congestion, the authors still consider the disappearance of the cells about the hepatic vein as a pressure atrophy. They do not mention the retrojection of bile among the etiological factors of hemorrhagic pancreatitis, and in

general this chapter is unsatisfactory. From the statement that absence of the pancreatic secretion interferes with the absorption of fatty substances in the intestines and in such cases the stools contain excess of undigested fat, one would hardly believe that this question is still in a very chaotic condition: again in their experience cancer of the head of the pancreas is rare, while it is generally considered to be rather frequent.

In the discussion of the ductless glands the distinction between the function of the parathyroids and thyroid glands is not sufficiently emphasized, nor is the relation of lesions of the adrenal to Addison's disease discussed to the extent our present knowledge would allow.

Briefly, the book is for the most part a clear exposition of the field of special pathology. While in scope it corresponds to many other books of the same order, it is well written, very well illustrated and will no doubt be of real use to the student of pathology, especially in those schools where the thorough system of the Edinburgh School has been adopted.

Arthritis Deformans: Comprising Rheumatoid Arthritis, Osteo-Arthritis and Spondylitis Deformans. By R. LLEWELLYN JONES, M. B. (Lond.). XIV, 365 pages. (*Bristol: John Wright & Sons Ltd., 1909.*)

There is so much obscurity connected with the subject of chronic arthritis that it is not an easy task to discuss it clearly and satisfactorily, and any work which does this is welcome. The majority of French and German writers on this subject cannot be said to have produced illuminating articles; they rather complicate the question than render it clear. Dr. Jones has given us a clear discussion of the subject. He has had excellent opportunities for the study of arthritic maladies. Living in Bath, to which for centuries invalids have repaired and where those suffering from arthritis go in large numbers, he has had the advantage of study in a hospital to which many patients with arthritis are admitted, and also of seeing many patients from elsewhere. The importance of increasing our knowledge of arthritis deformans is great. Not a disease which kills but one which cripples and incapacitates, every effort to learn more as to its nature and how to limit its power for harm must be welcomed. It may be said here that Dr. Jones has given us an excellent work on the subject.

After a historical review in which the ancient history of the disease is discussed he takes up the question of classification and supports strongly the view that under the heading arthritis deformans there are two distinct diseases, rheumatoid arthritis and osteo-arthritis. On this point there is great difference of opinion and recent articles illustrate this. Thus Garrod, in the article in Allbutt and Rolleston's System of Medicine, takes the same view, while McCrae, writing in the Osler System, takes the opposite position, that the various forms are differing manifestations of the one disease. Important support of this latter view is the frequency with which lesions characteristic of the two forms are found in the same patient. Dr. Jones considers this as the co-existence of two diseases, just as gout and arthritis deformans may be seen together, but this is exceedingly rare, while the commonness of the occurrence of the two forms of arthritis in the same patient is against this. There is evidence of the difficulty of the author's view in the discussion o. spondylitis deformans. For convenience of description this is discussed separately from rheumatoid arthritis and osteo-arthritis, but the author points out that "no sharp line can be drawn between it and the peripheral types of arthritis deformans, owing to the existence of endless intermediate or transitional forms." Dr. Jones, in discussing the etiology of spondylitis, speaks of the rigidity being "the result either of rheumatoid arthritis or osteo-arthritis." It is "the existence of endless intermediate or transitional forms" which renders it difficult to accept a sharp division of the whole group into two diseases.

Taking the view that there are two diseases the author naturally discusses them separately. The description of the symptoms is excellent throughout and especial attention, and most properly so, is given to the early symptoms. The importance of the careful study of these as an aid in the early diagnosis, so important for correct early treatment, cannot be overestimated. The question of treatment is well discussed and drug therapy is not unduly exalted.

The work is to be very heartily commended as probably the best discussion of the disease we have. The enormous literature on the subject has been well handled and it is a pleasure to see that the American literature has been carefully considered. The publishers have done their work well and the page and illustrations are alike good.

Tuberculosis. A Treatise by American Authors on its Etiology, Pathology, Frequency, Semeiology, Diagnosis, Prognosis, Prevention and Treatment. Edited by ARNOLD C. KLEBS, M. D. With three colored plates and 243 illustrations in text. (*New York and London: D. Appleton & Co., 1909.*)

There are a number of things about the book that do not please the reviewer. Others may not share his views and perhaps they are the result of personal peculiarity, but he neither expects nor seeks to elicit agreement. Most prominently disappointing is the arrangement of the bibliography. It would be much more valuable if each section were followed by its appropriate list of references and still more useful and convenient if they were added as footnotes at the bottom of the page. It is a source of constant annoyance to be obliged to turn to an alphabetical index when a judicious quotation or an important reference arouses a curiosity to know its source. After considerable investigation the reviewer is unable to say just what scope the bibliography was meant to have. It is more than a compilation of the works cited in the text and far less than anything like a complete list of tuberculosis literature. Apparently the plan was to include all publications by contributors to the volume whether they are quoted or not and to add such other references, important or otherwise, as are fortunate enough to have been mentioned. What practical use this scheme subserves it is difficult for one disinterested to understand. I take the instance of Knopf as an example merely because it represents most flagrantly what is meant. In the text he is directly cited not more than three or four times, yet in the bibliography there are fifty-six references to his writings. Now it is an important matter to have a complete list of the publications of Dr. Knopf and every student of tuberculosis literature will be duly grateful for the compilation. However, it does not seem clear why he is so honored when others, may I say equally prominent, and then name Koch, Behring, Römer, Turban, Möller, Bandelier, Röpke, Calmette, Arloing, Courmont, etc., are slighted.

As an addendum to each section there is a summary of the papers upon this particular subject presented at the International Congress, held in Washington, D. C. In no instance do these summaries add anything of importance or even of interest to what had previously been written. Did they contain such valuable additions, proper emphasis could be given them only by incorporation in the text. They lend the book a superficial air of unusual up-to-dateness which in substance they do not sustain.

The same remark might be applied to the brief introductory paragraphs written by prominent authorities. They add the glamor of a great name, but do not enhance the value of the book. We do not question the motive that led to their insertion, but the effect is to create the impression that they were added to give a stamp of authoritative co-operation and approval.

The notes by v. Pirquet on tuberculosis in childhood, while in

themselves excellent, seem scarcely appropriate as an introduction to the section on symptomatology and diagnosis. Still it is hard to say where they would be appropriate and so they might as well be placed here as elsewhere. Why they were added the editor tells us in an ingenious foot-note which, viewed from a certain standpoint, is not without humor. It might be questioned whether it is wise to exploit the general interest in our most recent American acquisition. I should certainly not dare to give the answer. A very celebrated artist has created a remarkable effect by filling a void in his most celebrated painting with two cherub heads which are popularly known as the "afterthought." v. Pirquet's article is an editorial "afterthought" and opinions might vary from one considering it an excellent but incongruous insertion to another looking upon it as a rare and brilliant inspiration.

The historical sketch is written in Osler's terse and pregnant manner. The etiology by Ravenel is notable for the author's restraint. Where so many controversial points are touched it is difficult for one interested in all their details to resist discursiveness. The matter is forcefully and judiciously presented. Fault can be found only with the heated attitude assumed towards Koch. Granting that he be wrong in his contention that bovine tuberculosis is a negligible factor in human infection, it would still be the part of valor to treat so distinguished an opponent with gentleness and consideration. Hektoen's chapter on pathological anatomy is remarkable. Astonishment ever increases at the amount of matter compressed in so small a space. It is not easy to read, does not flow smoothly, but there are few unnecessary words and not a superfluous sentence. Even subsidiary clauses often suggest a paragraph of important matter. Baldwin gives a good summary of the factors of immunity in tuberculosis, but to those interested in these problems, far too concise. On special phases of the subject he has elsewhere written far more thoroughly and entertainingly. A text-book naturally imposes restrictions which add great difficulty to a proper presentation of so intricate a subject.

The section by Klebs contains all the essential information on mortality and morbidity. Coleman reviews the special conditions relating to tuberculosis among the Negroes and Indians and Hutchins among the insane.

The section on symptomatology and diagnosis, the largest and most important in the book, is written by Minor. The arrangement is excellent and although it necessitates a certain amount of repetition, the importance of the points reiterated makes it impossible to emphasize them too frequently. The style is in places involved. As an example, reread the opening paragraph. Too frequent quotations detract somewhat from the force of personal views and could with benefit be omitted, or else added as foot-notes. They often force the impression of literary display which is evidently far from the author's purpose. It seems, however, unnecessary to seek the support of authority for everyday observations. I can make my meaning plainest by a quotation from Jules Lemaitre. Speaking of the oratory of Le Père Monsabré he writes: "The Père somewhat abuses citations from Saint Thomas. In his first conference he finds it necessary to invoke him to tell us that penitence is to the soul what medicine is to the body. The thought, however, is in no way extraordinary; the author should, I believe, have been able to hit upon it himself. One does not derange a saint for so little." Allowing that many of the authors quoted do not possess in medicine the same imposing authority that St. Thomas does in theology, the remarks are otherwise pertinent and we may justly say that the author somewhat abuses citations. Quite naturally, there are a number of minor preferences expressed with which all will not agree. I mention among others the value of the cyrtometer in diagnosis, the necessity of using the little finger as the plexímeter in apical percussion, and the superiority of fluoroscopy over radiography. We believe too that percussion resistance is felt earlier than the author admits. But these differences are trivial in the light of the general excellence of the presentation. The remarks on hemorrhage are strikingly good. It is refreshing to see inspection and percussion restored to their proper position of importance in early diagnosis. The chapter on the method of examination could not be improved. Indeed, all in all, it is the most satisfactory treatment of the subject in English with which the reviewer is acquainted.

Individual prophylaxis is satisfactorily reviewed by Baldwin. Knopf treats public prophylactic measures in his usual full and enthusiastic manner. His caution that the popular lecture should not exceed three-quarters of an hour may provoke a smile.

Brown has written a most comprehensive chapter on specific treatment and one which shows at every point his intimate acquaintance with all phases of the subject. He assumes a moderate and a very open attitude towards tuberculin. The fundamental principles of treatment are thoroughly reviewed, although the theoretical discussions lack precision and clearness. Some of the statements it is difficult to reconcile. He says Wright's method is a step in the proper direction and on the following page that in practice it may be disregarded. He asserts that the results of tuberculin immunization have never been as striking as those attained by small repeated doses of tuberculin, while later he admits that many have not been able to substantiate these results. While granting that there is little difference between the action of the various tuberculins, he advances reasons why a mixture of B. F. and B. E. is to be preferred. This author also has a tendency to abuse citations.

Webb gives a brief but excellent account of the vaccine treatment of secondary infections.

The chapter on the hygienic, dietetic and symptomatic treatment is far too brief. It occupies but forty pages, while climate is allowed fifty-seven. It is a good practical exposition, but diet particularly lacks the scientific detail that a modern treatise should give. Klebs has a good account of the management and construction of sanatoria.

The physiology of climate is well presented by Sewall, but the knowledge gained will add little in its practical application. Barlow takes a refreshingly moderate view about the value of climatic therapeutics.

The whole subject of surgical tuberculosis is written by Freeman and McArthur. The consideration of the individual subjects is necessarily brief, and indeed in proportion to their importance some of them disappointingly brief, notably tuberculous peritonitis. A separate section on serous membrane tuberculosis would be desirable.

The list of prescriptions at the end of the book should, if important enough, be given their proper position in the chapter dealing with treatment. Loosely thrown together they are of little value and they may do a great deal of harm. They are certainly not written in the spirit of modern therapeutics.

In conclusion, a word about the impression the book as a whole makes. The work of the contributors is for the most part well done and they have succeeded in producing a truly valuable work. With such alterations as the foregoing remarks suggest, the reviewer would not hesitate to pronounce it excellent. L. H.

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